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NEURAL MECHANISMS OF HEARING: SOME EXPERIMENTAL STUDIES OF THE AUDITORY NERVOUS SYSTEM.[1*]

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Chicago, Ill.

I. Introduction.

Theories of the neurophysiological basis of hearing have been based primarily upon results of anatomical and physiological investigations. A complex system of neural tracts and nuclei has been described in some detail, and observations have been made on the flow and interaction of nerve impulses in this system. Although both kinds of evidence, anatomical and neurophysiological, are still incomplete, they have been sufficient for the formulation of fairly elaborate hypotheses as to how such a system might mediate "hearing"—the name we usually use to refer to behavior which is initiated or guided by sound stimuli.

As a further step in the development of a theory of hearing, we need another kind of evidence, the kind that comes from experiments which are sometimes labeled psychophysiological, or more simply, "behavioral." These are experiments

[1]The experiments cited in this paper were supported in part by the Office of Naval Research, Contract N6 ori-20, T.O. XXIV, ONR Project NR 140 608.

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in which the nervous system is manipulated (*e.g.*, by section of a tract or ablation of a nucleus) and the effect upon some auditory discrimination is measured.

In the present paper, two examples will be given to illustrate *1.* how psychophysiological experiments are suggested by the hypotheses formulated from anatomical and neurophysiological evidence, and *2.* how the results of the experiments may lead to revision of theory concerning the neural mechanism of hearing.

II. The Discrimination of Changes in Frequency.

From a number of experiments there is rather clear evidence that the cochlea is projected upon the cerebral cortex in a systematic fashion (Woolsey and Walzl, 1942; Licklider, 1941; Tunturi, 1944, 1950).

In Fig. 1 maps of the auditory areas of the cortex of the cat and dog are shown. Woolsey and Walzl mapped the auditory areas of the cat in the following manner: In an anesthetized animal, the cochlea was opened so as to expose the nerve endings along the spiral lamina. At different points from base to apex, these endings were stimulated by weak electric shock. At the same time the potentials evoked at the cortex were recorded. In this way two auditory areas (see AI and AII, Fig. 1) were plotted. In AI, the base of the cochlea was projected anteriorly in the ectosylvian gyrus and the apex, posteriorly. In AII, which extended from the anterior ectosylvian across the pseudosylvian and into the posterior ectosylvian gyrus, the apex of the cochlea was projected anteriorly and the base, posteriorly. In the original experiment, area AI was shown as extending to the postsylvian sulcus. Later, Rose and Woolsey (1949), on the basis of both cytoarchitectonic and electrophysiological results, produced the map as shown in Fig. 1A, in which a third, posterior ectosylvian area is shown in addition to areas I and II.

In a series of studies on the dog, Tunturi obtained results which may be summarized briefly by the map of Fig. 1B. He, also, used an evoked potential technique but stimulated the

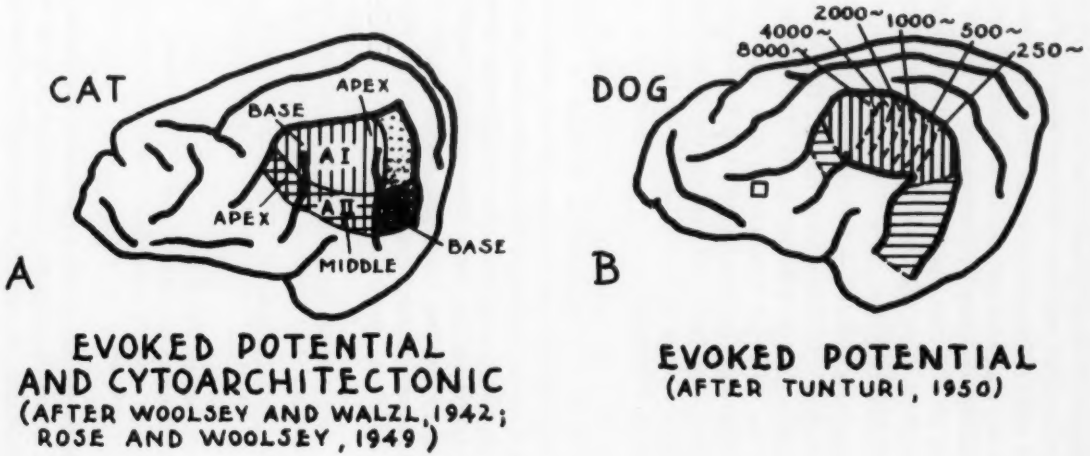

Fig. 1. Maps of the auditory cortex.

intact cochlea with tonal pulses instead of applying electrical stimuli to the nerve endings. Tunturi described an auditory area in the middle ectosylvian gyrus (vertical lines) in which the points of maximal potentials aroused by tonal stimuli could be plotted as shown; frequencies at octave steps fall along bands about 2 mm. apart on the cortex. Potentials were also elicited from other regions of the ectosylvian gyrus (horizontal lines and square).

The finding of a rather precise representation of the cochlea upon the cortex (Woolsey and Walzl), the evidence from many experiments that different parts of the cochlea respond maximally to different frequencies, and the evidence that tones of different frequencies affect different parts of the auditory area (Tunturi), all lead to the hypothesis that the auditory cortex plays an essential rôle in the discrimination of changes in frequency.

To check this hypothesis, an experiment was done in which cats were trained to respond to changes in frequency, the auditory areas of the cortex were ablated, and postoperative tests were made to determine the effects of the ablation (Butler and Neff, 1950). An avoidance conditioning procedure was used to obtain hearing tests. The animals were trained to avoid shock by moving from one compartment of a double grill box to another; the signal for a response was a change in frequency of an interrupted tone.

TABLE I.—FREQUENCY DISCRIMINATION BEFORE AND AFTER BILATERAL ABLATION OF THE AUDITORY CORTEX.

	Preoperative			Postoperative		
	Standard	Comparison	DL	Standard	Comparison	DL
Cat 1	200 c.p.s.	2000	—	200	200-205	5
Cat 2	200 c.p.s.	300-250	—	200	250	—
Cat 3	200 c.p.s.	250-205	5	200	—	—

From Butler and Neff.

Preliminary results of this experiment are shown in Table I. Cat 1 was trained to respond to a large change in frequency—200 to 2,000 c.p.s. No attempt was made before operation to

determine the difference limen (DL), that is, the smallest discriminable frequency change. After bilateral ablation of auditory areas I and II of Woolsey and Walzl, the cat relearned the 200-2,000 c.p.s. discrimination, and, upon further training, was able to make a discrimination of 200-250 c.p.s. and finally 200-205 c.p.s.

Cat 2, trained preoperatively to a difference of 50 c.p.s., was able to perform at the same level postoperatively. The preoperative record of Cat 3 is shown for comparison with Cat 1. A difference limen of 5 c.p.s. was found for Cat 3 preoperatively. This is the same as the postoperative difference limen for Cat 1.

On the basis of these results, which must be considered tentative until further experiments are completed, the hypothesis that the auditory areas of the cat are essential for discrimination of frequency must be rejected.

III. Localization of Sound in Space.

In discrimination requiring the localization of sound in space, the interaction of neural impulses initiated at the two ears is of primary importance. Anatomical evidence suggests that this interaction may take place at any of several levels of the nervous system — cortex, inferior colliculus, superior olivary complex, primary cochlear nucleus, or perhaps even at the level of the spiral cochlear ganglion (see *e.g.*, Ades, 1944; Rasmussen, 1946). The effect of interaction has been measured by electrophysiological methods at the cortical level (Rosenzweig, 1950), at the inferior colliculus (Ades and Brookhart, 1950), and at the cochlea (Galambos, Rosenblith and Rosenzweig).

Neither the anatomical nor electrophysiological evidence answers the question: At what level can the afferent signal lead to the appropriate motor behavior, the localizing response to a sound stimulus? Again behavioral evidence is necessary.

A series of experiments has been done in which the ability of cats to localize sound has been measured before and after ablation of parts of the auditory nervous system (Neff and

Yela, 1948; Arnott and Neff, 1950; Neff, Arnott and Fisher, 1950). The animals were trained to approach and open the door of one of two boxes in order to obtain food reward. The correct choice on a given trial was indicated by a sound stimulus (buzzer) placed behind one of the boxes. The angle between successive positions of the buzzer as measured from a starting cage could be varied from 180° to 0°.

No change in ability to localize was found after unilateral ablation of the auditory areas I and II. An unmistakable change in performance appeared, however, after bilateral removal of these areas. Normal animals are able to make correct discriminations when the angle between successive positions of the sound stimulus is as small as five degrees. Animals without auditory cortex reach the same criterion of performance only for angles of 40 degrees or greater. The behavior of operated animals was also more variable than that of normals. A high percentage of correct responses was not maintained over a long period of time even when the angle to be discriminated was very large.

It is concluded that the auditory cortex is not essential for discriminations which involve the localization of sound in space; it is necessary, however, for the most effective utilization of sound cues in guiding such behavior.

The experiments cited above were done in collaboration with R. A. Butler, G. P. Arnott, J. D. Fisher and M. Yela.

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HEADACHE AND AUTONOMIC IMBALANCE.*

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Headache is one of the commonest complaints encountered in medical practice. It is usually the symptom in a syndrome; therefore, a physical examination and a complete history are necessary for differential diagnosis. Vascular headaches, most usually typed as migraine, because of a definite mechanistic pattern, can be classified as a definite clinical entity. Lennox[1] has conservatively estimated that there are six million migraine or migraine type sufferers in the United States, which probably includes the entire classification of vascular headaches. Others estimate as many as ten million. Vascular headaches include classical migraine, tension headaches, migraine equivalents, histamine cephalgias, and a group of unclassified vascular headaches, all of which challenge the diagnostic ability of the practicing physician.

The acute attack of vascular headache is now adequately controlled with such vasoconstrictors as ergotamine tartrate, dihydroergotamine, and oral ergotamine tartrate with caffeine alkaloid. Horton,[2] Alvarez,[3] Ryan,[4] Hansel,[5] Friedman,[6] Wolff[7] and Hilsinger,[8] among others, have reported excellent results ranging from 80 to 90 per cent with all or some of these drugs. It must be pointed out that up to the present these drugs are only effective in the acute attack. Employing them, especially Cafergot, for interval therapy will not reduce the incidence or frequency of attacks.

It is the purpose of this paper to report the use of a drug which, in our hands, has been found effective in prolonging

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the quiescent period between acute attacks of vascular headaches. The need of such therapy is indicated because:

1. vascular attacks do not always respond promptly to vasoconstrictor medication.
2. the taking of an ergot preparation over long periods is not always desirable,
3. some of the medications employed result in undesirable side effects as nausea, cramps, vomiting, etc. Such effects present psychologic hazards to the patients so that they become somewhat resistant to medication, and subsequent attacks are more difficult to control.

Friedman[6] has stressed the personality and emotional conflict aspect of these headaches. The most common precipitating factors, *i.e.*, worry, fear, anger, etc., place strain on the autonomic nervous system. For this reason, interval treatment must be aimed at removing these, usually by psychotherapeutic means. Sufficient rest and removal of undesirable environment stimuli has been recommended and found effective.

Drug therapy is also indicated if it can be directed towards dampening the responses of the autonomic nervous system. The use of such a drug enhances greatly the psychotherapeutic measures and enables the patient to adjust his living in a quieter and easier fashion. We have found that the following combination fulfills this purpose:*

0.3 mg. ergotamine tartrate—sympathetic inhibitor
0.1 mg. Bellafoline—parasympathetic inhibitor
20 mg. (1/3 gr.) phenobarbital—central sedation

Rothlin,[9] Jores and Goyert,[10] and Tod and Jones[11] reported on the rationale for and the results with this combination on quieting the autonomic nervous system. This particular combination apparently acts in synergism and is effective in reducing the intensity of nerve response to stimulating experi-

*Manufactured under the trade name of Bellergal, Sandoz.

ences. Moore,[12] Slagel,[13] Karnosh and Zucker,[14] Harris[15] and Nelson[16] all reported beneficial clinical results from its use.

RESULTS.

Twenty-five patients are reported in this series. Admittedly, a larger series would be desirable but, because of the criteria used in selecting these patients, this was difficult to attain. Only those patients with frequently recurring attacks who did not respond to the more common vasoconstrictor drug therapy, or those who complained of pronounced side effects from these drugs, were selected.

Dosage of this drug must be adjusted to the individual needs of each patient. The usual recommended dosage is four tablets per day — one 15 minutes before each meal and one at bedtime. This dose can be increased to five or six tablets when these individuals are under greater stress. The maximum dose is given at the beginning of the therapeutic regimen and then gradually is decreased as improvements appear. Finally, some individuals will require only one tablet daily as a maintenance dose. Occasionally the larger dose may produce some drowsiness, in which case dosage must be reduced. Although Slagle[13] reports no deleterious side effects from this combination after prolonged use in 217 patients, prolonged use of any sedative is usually not recommended. Patients may be advised, therefore, to interrupt treatment two days out of every week, especially when maintained on a high (four to six tablets) dosage level. A similar recommendation is made by Harris[15] who suggests interrupting therapy with this combination one week out of four. This recommendation is made to avoid any possibility of habituation caused by the phenobarbital component.

Clinical results and outstanding case histories follow. Results for the entire series can best be seen in Table 1. A few case histories were summarized in order to show the variety in dosage. This points out again the necessity of individualized adjustment in order to obtain maximum effect from this type of therapy.

Case 2: G. M., female, age 23. Typical migraine since puberty with complete cessation during pregnancy. The migraine attacks occurred every two or three weeks. Cafergot and DHE-45 gave no results. One Bellergal was given 15 minutes before each meal and another was given at bedtime several days before the impending attack. The patient is now completing her twenty-fourth week with one mild migraine attack, and for the last few weeks has been maintained on one tablet per day.

Case 6: M. B., female, age 49. This case was diagnosed as histaminic cephalgia. The headaches occurred every three to four days. For the past year the frequency of attacks has been notably reduced as a result of histamine desensitization; however, the patient complained of a tension type headache which occurred every three to four days to one per month. Bellergal therapy was initiated and the patient is now completing her fifteenth week completely free of all headaches.

Case 7: H. G., male, age 29. Gave a history of grand mal epilepsy which has been completely controlled for the past two years with 3 gr. of phenobarbital daily; however, he continued to suffer with a tension type headache which formerly followed his epileptic seizures. These incapacitating headaches occurred weekly and would last for from four to 12 hours. Cafergot gave poor results. Nicotinic acid gave moderate relief. Bellergal therapy was initiated along with nicotinic acid and the patient has been completely free of headaches for the past 14 weeks.

Case 10: M. M., female, age 68. Typical migraine since puberty. Headaches increased in severity since menopause 16 years ago. The headaches occurred regularly at two-week intervals, lasting for 48 hours, during which time the patient had to go to bed. Cafergot, Gynergen and DHE-45 gave no relief. Nicotinic acid gave mild relief. Bellergal therapy was initiated. One tablet was taken 15 minutes before each meal. Another tablet was taken at bedtime several days before the impending headache. The patient is now concluding her eighteenth week with one attack of migraine.

Case 11: P. G., male, age 37. Complained of a dull throbbing unilateral incapacitating headache. The patient had no aura and complained of nausea during the attack. Headaches were irregular in frequency, occurring once in three to four days to one every two weeks. The headaches lasted from 24 to 30 hours. The patient gave a family history of headaches. Case was diagnosed as a migraine equivalent. Cafergot, DHE-45, codeine and nicotinic acid were of little or no value. Interval treatment with phenobarbital was also of no help. The patient actually lived in fear of these headaches. Bellergal therapy of 1 q.i.d. was initiated and the patient is completing his seventeenth week with one very mild headache.

Case 14: L. G., male, age 33. Frequently recurring headaches since 30 years of age. Headaches were of comparatively short duration and occurred during early morning. The case was diagnosed as a typical histaminic cephalgia. Nicotinic acid gave moderate results, and good results were obtained with Cafergot; however, because of the frequency of the attack, it was felt that this patient was receiving too large an amount of ergotamine tartrate. The patient was placed on one Bellergal tablet 15 minutes before each meal, and he is completing his ninth week completely free of headaches. Of special interest is the fact that after about the fourth week the patient voluntarily offered the information that the symptoms from his duodenal ulcer have also been greatly relieved and that he had stopped going to his "stomach doctor."

Case 17: L. B., female, age 55. Complained of headaches since puberty. Intelligent executive secretary. This case was diagnosed as a typical migraine. The headaches were so severe that she had to stay home three to four days each month. The headaches occurred every one to two weeks. Bellergal therapy was initiated and, to date, the patient has had two mild attacks of no consequence in 26 weeks.

Case 18: R. G., male, age 48. Tension headache and nasal allergy. Cafergot, DHE-45 and interval phenobarbital and antihistaminics gave poor results. Headaches occurred every three to four days, to one every two weeks. Bellergal, 1 t.i.d., was given, and 1 q.i.d. on days when the patient encountered added strain. At this time the patient has been completely free of headaches for 14 weeks. During the last several weeks the patient has adjusted his own dosage so that no medication is taken on certain days, with a maximum of four on others.

TABLE I. CLINICAL RESULTS.

	Name	Age	Sex	Diagnosis	Frequency of Attack	Length of Attack (Hours)	Duration of Bellergal Therapy (Weeks)	Number of Attacks since Bellergal Therapy
1.	L.F.	37	F	Tension	1/3-4 days 1/1-2 wks.	12-48	17	None
2.	G.M.	23	F	Migraine	1/2-3 wks.	48	24	1 Mild
3.	J.M.	32	M	Tension	1/2-3 wks.	8-12	12	None
4.	E.V.	45	M	Migraine Variant	1/2-3 wks.	4-8	14	2 Mild
5.	T.B.	40	F	Tension	1/2-3 days	8-24	14	1
6.	V.B.	49	F	Tension	1/4-5 days	8-12	15	None
7.	H.G.	29	M	Tension	1/wk.	4-12	14	None
8.	R.S.	46	M	Tension	1/2 wks.	8-12	29	4
9.	D.M.	35	M	Tension	1/2-3 wks.	12-24	8	1 Mild
10.	M.M.	68	F	Migraine	1/2 wks.	48	18	1
11.	P.G.	37	M	Migraine Equiv.	1/3-4 days 1/2 wks.	24-30	17	1 Mild
12.	N.R.	72	F	Hist. Ceph.	1/wk. variable	1-2	10	None
13.	C.R.	37	F	Tension	2/wk.	8-24	8	2/wk. Possible slight improvement.

TABLE I. CLINICAL RESULTS (Cont.).

	Name	Age	Sex	Diagnosis	Frequency of Attack	Length of Attack (Hours)	Duration of Bellergal Therapy (Weeks)	Number of Attacks since Bellergal Therapy
14.	L.G.	33	M	Atypical Hist. Ceph.	1/3-4 days variable	1-2	9	1 Mild
15.	M.S.	36	F	Tension	1/wk.	24	11	None
16.	M.Z.	28	F	Tension	1/1-2 wks.	8-10	15	None
17.	L.B.	55	F	Migraine	1/4 days 1/2 wks.	12	26	2 Mild
18.	R.G.	48	M	Tension	1/5 days 1/2 wks.	24	14	None
19.	E.H.	37	M	Tension	1/2-2 wks.	12-24	24	2
20.	L.M.	35	F	Tension	1/2-3 days	2-8	16	4
21.	M.T.	56	F	Tension	1/2 wks.	4-8	10	None
22.	L.G.	51	F	Tension	1/1-2 wks.	8-12	8	None
23.	L.S.	44	F	Tension-accentuated at menstruation	1/2 wks.	4-12	11	1/2 wks. No results.
24.	C.W.	50	F	Tension	1/wk.	8-12	17	4
25.	M.E.	26	F	Migraine variant	1/2 wks.	8-12	11	1

SUMMARY.

1. Autonomic imbalance is a major contributing factor in vascular headache.

2. During the acute attack, good results may be obtained with ergotamine tartrate, dihydroergotamine or "Cafergot."

3. Rest and removal of undesirable environmental stimuli is the preferred method of prolonging the quiescent period of frequently recurring vascular headache. Where this is im-

practical, a drug which will dampen the effects of the undesirable nerve impulses to the autonomic nervous system is recommended. A combination consisting of ergotamine tartrate (sympathetic inhibitor), bellafoline (parasympathetic inhibitor) and phenobarbital (central and subcortical sedative), (Bellergal), has been found to be effective for this purpose.

TABLE II. SUMMARY OF CLINICAL RESULTS.

TYPE HEADACHE	NO. CASES	EXCELLENT*	GOOD**	POOR***
Migraine	6	4	2	
Tension	17	11	1	5
Histaminic Cephalgia	2	2		
TOTAL	25	17	3	5
% Results		68%	12%	20%

*Complete relief, or a maximum of one attack.
**Noted reduction in number of attacks.
***Little or no relief.

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OBSERVATIONS IN TREATING SEVEN CASES OF CHOANAL ATRESIA BY THE TRANSPALATINE APPROACH.*†

HAROLD OWENS, M.D.,
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Congenital choanal atresia is thought to be due to the interrupted or arrested development of tissues in the choanal region. Boyd[2] states the condition is not really an atresia, because it results from failure of normally occurring embryonic membrane to disappear.

I do not intend to include a detailed discussion of the embryology involved in choanal atresia. This has been done well many times by Anderson,[1] Boyd, Colver,[5] Wright, Shambaugh and Green,[16] and others. In simplest terms it is now thought to be a malformation of the nasobuccal membrane or the buccopharyngeal membrane. The amount of mesodermal tissue which remains between the layers determines whether the obstruction is to be membranous or bony. Swartz and Isaacs[15] have demonstrated islands of cartilage in the obstructing bone. Boyd states that if the obstruction came from membrane it would be membranous bone and not contain cartilage. This observation brings up the third etiologic possibility of medial overgrowth of the vertical and horizontal palatal processes as a cause of the obstruction. This last observation may explain some of the consistent findings at surgery which I will relate later.

Ever since choanal atresia was described by Otto,[12] in 1829, the treatment of this condition has presented a challenge. The

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first case treated by Emmert,[7] in 1851, was relieved by puncturing with a trochar. Similar methods are still being used at the present time, usually through the intranasal route. Burrs, electrocautery, chisels and ronguers have all been added to the armamentarium. The condition is not too common, Anderson reporting only six cases at the Mayo Clinic during a 30-year period and Kazanjian[9] reported only 10 cases for a 16-year period at the Massachusetts Eye and Ear Infirmary. Not many men have had a series adequate enough to evaluate results. Many single case reports are in the literature. Surgical correction has been poor because of cicatricial contraction of the new opening. This was particularly true when the procedure was attempted intranasally. To circumvent this, other methods were devised. Brunk,[4] in 1909, attempted a transpalatine approach, but it was a failure. Blair[3] performed an ingenious transpalatine operation in 1931, in which he manipulated mucosal flaps and resutured them to preserve the respiratory mucosa. This necessitated a rather extensive removal of normal tissue.

Lorenz Ruddy[13] presented a paper describing a transpalatine approach in 1945. He thought at first his idea was original, but on reviewing the literature he unearthed several other reports on the palatine approach, mostly in the foreign literature and then describing only one case. I advise anyone interested in the details of the various methods to study Ruddy's fine article.

Just before Ruddy's report I had been toying with a bilateral bony atresia which I had opened as an emergency by pushing a trochar through the obstruction by the intranasal route and then inserting acrylic tubes. The care necessary by me, and more so by the mother, as well as the long convalescence with the tubes in place, left much to be desired. This new approach by Ruddy seemed ideal to me and was carried out to a very successful conclusion.

Although Ruddy's surgical approach was not the first transpalatine procedure to be described, it was a much easier one and sacrificed less tissue. In any event, the article seems to

have stimulated others to use the method, as several papers have appeared since, with some modifications.

I, too, have modified it slightly, but basically it is Ruddy's operation; however, it has been my good fortune to operate on seven successful cases, and I feel that this warrants stumping for the procedure.

The approach offers advantages of attacking the problem under direct vision, and not by blind mutilating surgery. To illustrate this I might cite one case of a four-year-old boy with unilateral choanal atresia, first treated by the intranasal route with perforator and rasp. This opening soon closed and I did the second operation by a transpalatine approach. Under direct vision the posterior nasal fossa was one solid band of scar tissue extending forward to almost the middle of the nose; certainly over a much greater area than the original obstruction. With all this scarring even the transpalatine operation was not at first successful, and the palate was reopened and a skin graft was placed around a central sponge core. This second procedure insured the patency of the choana. I feel that observation of this condition well illustrates why so many done by the intranasal route are doomed to failure. Passing instruments through the narrow passage in the obstructed side of the nose creates raw surfaces for synechiae to form over in much greater area than the original obstruction.

The transpalatine approach gives greater insight into the problem at hand and in doing seven cases certain things became consistently evident: first, the nasal cavity on the obstructed side, especially posteriorly, is quite narrow (see Fig. 1-A). This is not due to a deviated septum but to a crowding of the lateral wall medially with a sweeping in of the lateral and posterior pharyngeal walls, thus shortening the anterior-posterior plane in the pharynx. Stinson's[14] article, in 1932, is the only one I have noted which mentions this finding, although Wright, Shambaugh and Green selected a transantral approach because of the narrowing of the nasal cavity on the obstructed side. Because of the above findings,

the obstructing membrane is angulated to about a ten-four o'clock position when observing it through the palate (see Fig. 1-B). This is not shown in most drawings where the membrane is usually shown in the transverse position. It is this shortening of the lateral and posterior wall of the

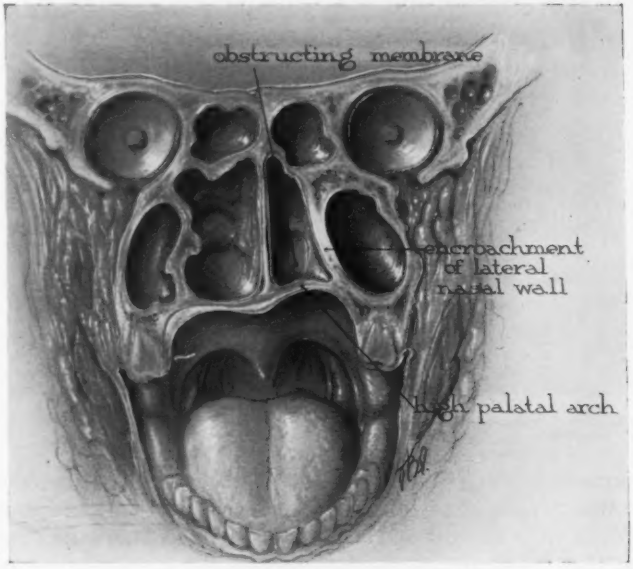

Fig. 1-A. Coronal view at the level of the choanal obstruction on the left. Note the palatal elevation and the narrow nasal cavity.

pharynx on the obstructed side, with the crowding of the lateral nasal wall, which makes removal of part of the vomer necessary for success of the operation. Because of the above deformity, even after operation, when probing the opening, the probe will follow downward and medially to drop into the pharynx instead of straight back as in the case of a normal choana. A higher palatal arch on the obstructed side is practically always present.

Why such deformities exist is not clear. The sphenoid body and medial plates of the pterygoid processes along with the septum and palatal bone form the choana. It has been shown by McGovern[11] that sinus and accessory auditory development

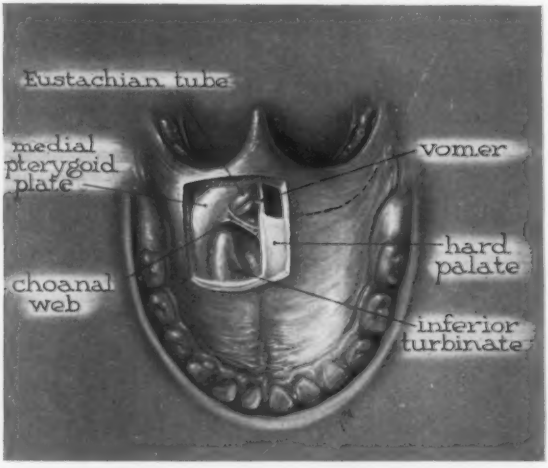

Fig. 1-B. Exploded view showing the crowding of the lateral nasal wall and the shortened nasopharyngeal cavity on the obstructed side.

are normal. An X-ray of one of my cases (see Fig. 2) shows normal development of the sinuses even though the nasal fossa is quite narrow on the obstructed side. This might suggest that the theory of medial overgrowth of the vertical and horizontal palatal processes as an etiologic factor is a good one; however, as the maxilla, sinuses and Eustachian tube are known to be well developed, it would seem to me that lack of development could be a mechanical one due to the restriction of the obstructing web, whether bony or membranous. For this reason, it would seem a logical argument to operate on these cases early. From a technical standpoint this can be done easily at a very early age by the transpalatine approach. This brings up the argument that removal of so much tissue may in itself retard development. In answer to this, I must state that a year after surgery I have palpated the posterior

border of the hard palate, and it is hard to discern any difference in the level of the operated and unoperated sides. Removal of a good portion of the vomer doesn't seem to interfere with development of the nose as sometimes supposed, and oral surgeons not infrequently remove a section of the

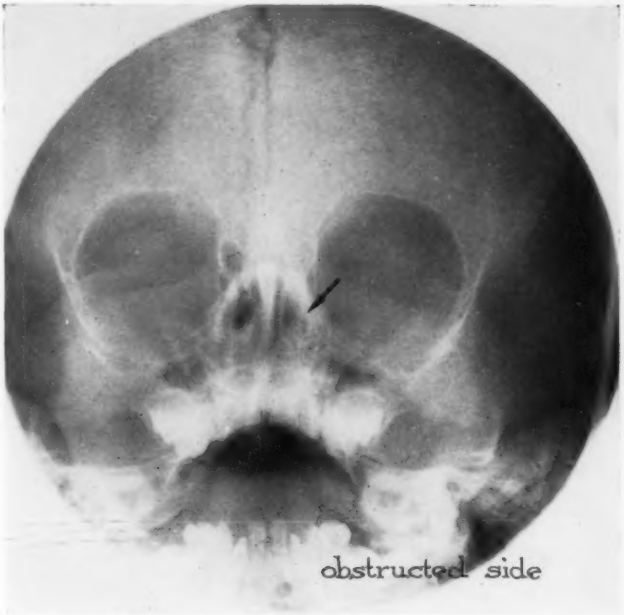

Fig. 2. X-ray showing choanal obstruction on the left side.

vomer to force back the premaxilla in cases of harelip. My first case, done at six months, and now five years postoperative, has normal nasal and facial development.

Many otolaryngologists are loath to enter the palate, but I assure you it is an easy and safe procedure. For those of you who are not familiar with the technique or have forgotten, I will describe it and show you my modifications.

Ether anesthesia is given through an intranasal catheter on the obstructed side. An incision is made in a semicircular manner from just inside the second molar tooth well forward to just a centimeter in front of Scarpa's foramen (see Fig. 3). It will be seen that this incision extends much farther

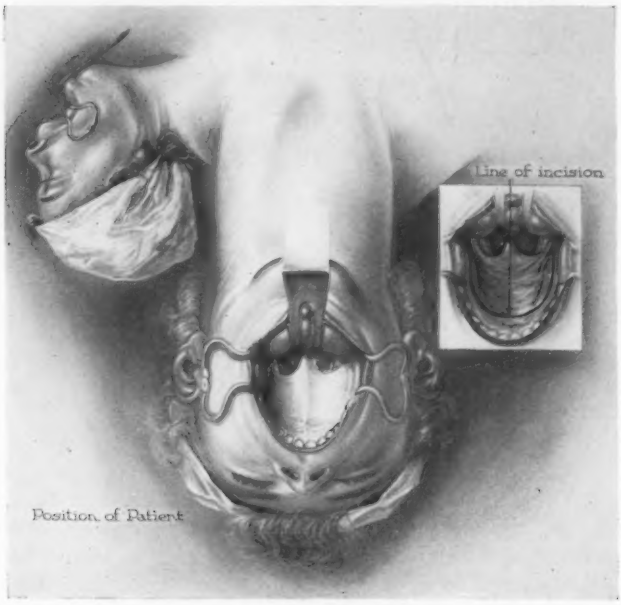

Fig. 3. The procedure is done standing at the head of the patient and the incision starts from the second molar area and extends around to 1 cm. posterior to Scarpa's foramen.

forward than Ruddy's original incision and also more laterally to include the greater palatine vessels in the flap. The incision is carried down to bone. This larger flap seems to allow for better healing, as all the cases have healed by primary union in a few days without slough. Inclusion of the arteries in the flap insures a good blood supply. This flap also does away with any fear of resulting perforation if one finds it

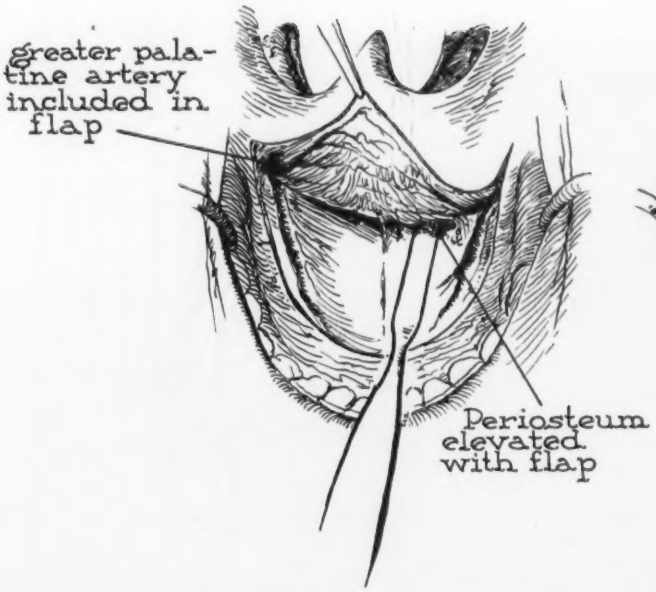

Fig. 4-A. Elevation of palatal flap. Note vessels in relief on under surface of the flap.

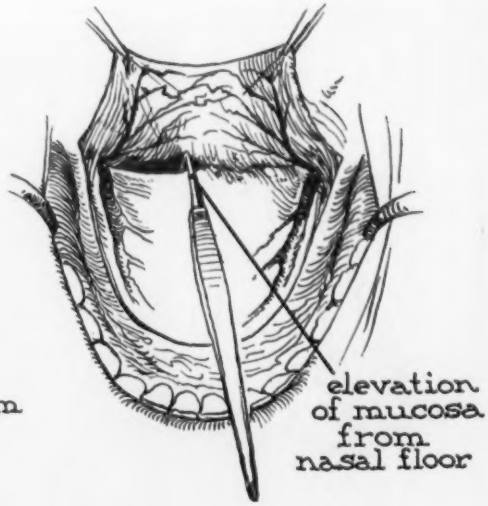

Fig. 4-B. Right angle Freer elevator freeing mucosa on the nasal floor.

Fig. 5-A. Palatine bone removed with Hajek rongeur.

Fig. 5-B. Palatine bone removed, but nasal mucosa intact. Dotted line indicates site of choanal obstruction.

necessary to remove much of the hard palate. I like this safety factor.

The flap is then elevated with the periosteum from the palate back to the posterior border of the hard palate (see Fig. 4-A). During this procedure the greater palatine vessels are lifted from their groove in the hard palate and are seen in relief on the under surface of the flap. When this is done the whole flap retracts back, evidently due to the pull of the muscles of the soft palate. With a right angled Freer knife or similar instrument the mucous membrane of the floor of

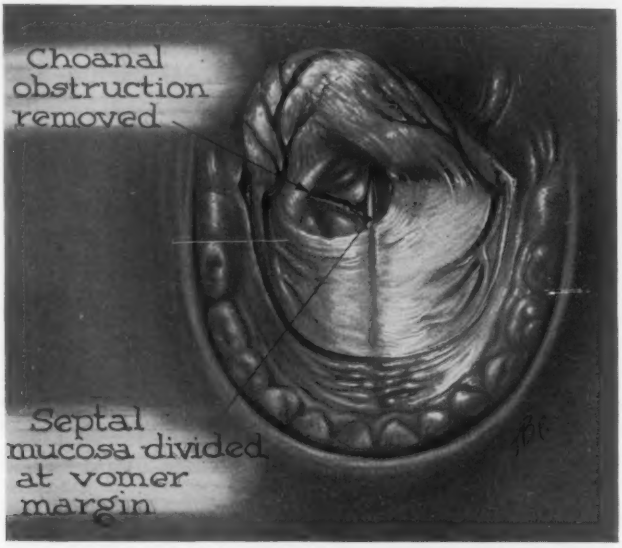

Fig. 6. Choanal obstruction removed and septal mucosa divided at vomer margin.

the nose is reflected forward to the obstruction (see Fig. 4-B). The palatal bone is removed with Hajek punch rongeurs forward to the site of obstruction (see Fig. 5-A). If the obstruction is membranous one can continue the forward removal of the palate until good exposure is obtained (see Fig. 5-B). If it is bony, then it is better to remove the palate

ahead of the obstruction with a gouge so that one gets a view on either side of the obstruction, as suggested by Ruddy.

After viewing obstructing plate one proceeds to bite it out clean to the margins with rongeurs (see Fig. 6). Goodhill[8] recently did a case by the transpalatine approach and he used a burr to take down some of the lateral wall in the region of

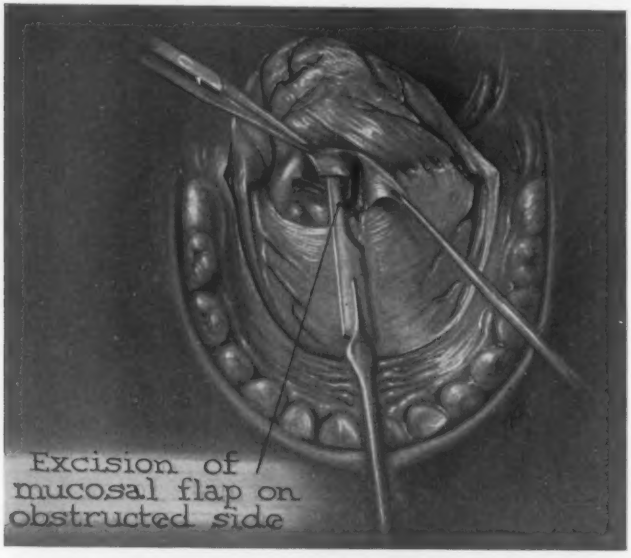

Fig. 7. Septal mucosa flaps separated and vomer bone removed. The septal mucosa on the obstructed side is being removed.

the pterygoid bone. This appears to be a good adjunct in the technique to increase the opening by dealing with the crowding lateral wall. I intend to try this on my next case.

Now the septum has to be dealt with. I carry my removal of the palate slightly over the midline so as to expose the vomer attachment to the palatine crest. Here again, I vary the technique of Ruddy slightly. Before cutting the septum, I incise the mucosa along the posterior border of the vomer and then separate the mucosa on either side as in a sub-

mucous resection. The bony septum is then removed as much as is deemed necessary for an adequate opening. The mucosal flap on the operated side is then removed and the opposite flap is severed at the anterior and posterior margins of the septectomy (see Fig. 7). This creates a flap which can be laid

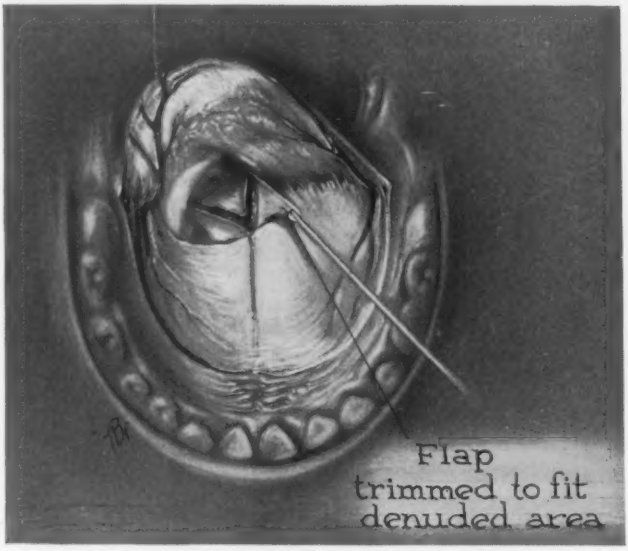

Fig. 8. Mucosal flap developed to be swung over covering denuded area.

over, covering the raw surface at the obstructoin site (see Fig. 8). If necessary the mucosa around the obstruction site can be denuded some to receive the raw surface of the flap. This flap is held in place with interrupted 00000 chromic catgut (see Fig. 9). Thus, about one-half of the opening is now lined with new mucosa.

All bleeding is controlled and then the palatine mucosal flap is replaced with interrupted 000 silk sutures, working from

the posterior poles forward (see Fig. 10). Slight elevation of the adherent anterior margins of the incision facilitates suturing.

No postoperative packing or tubing is necessary and sutures can be removed from the palate the fourth postoperative day.

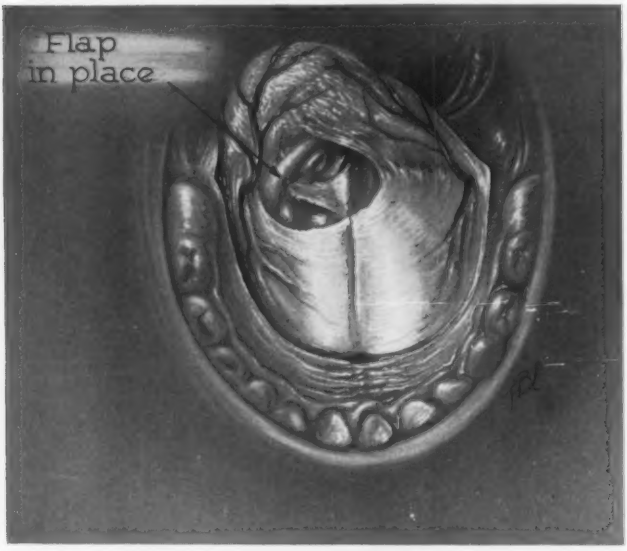

Fig. 9. Septal mucosa flap sutured in place.

Postoperative care consists of keeping the opening patent and free from crusts. The patency is tested by passing suitable dilators.

Dolowitz and Honey[6] described an ingenious way of handling a unilateral bony atresia by a palatine trephine with a submucous resection of the bony obstruction and then handling the mucosal flaps intranasally. The case of bony atresia which I treated was bilateral and needed to be treated in infancy. I feel that it would be difficult to handle any flaps at this age by the intranasal route. It would seem to me that a membranous atresia would also offer difficulty in splitting it to produce

a raw surface on the flaps. I am not convinced that flaps help physiologically, because I wonder if correct ciliary action is maintained when we maneuver flaps in different directions or if there are cilia on the surfaces of the obstructing web.

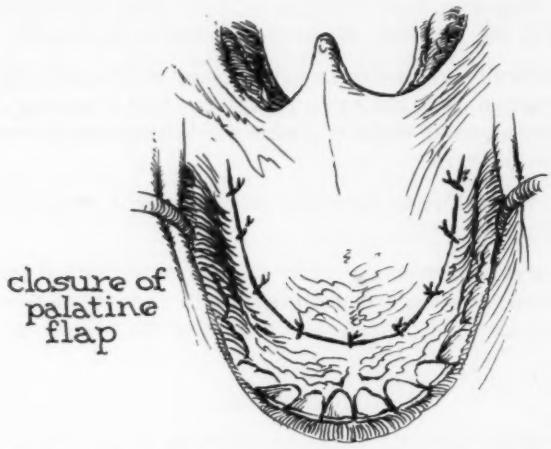

Fig. 10. Palatine flap replaced with interrupted silk sutures.

McGovern observed material passed into the pharynx on the obstructed side after surgery without using mucosal flaps. This was confirmed by Wright *et al.* When flaps are used, there is no doubt that it minimizes cicatricial contraction.

It is interesting to note that Libensohn[10] states that 90 per cent of such obstructions are osseous. Five of seven cases, or 71.5 per cent, in my series were osseous.

My youngest case was, of course, the bilateral bony atresia which I did on a patient six months of age, after first doing intranasal punctures at three weeks of age. My oldest case was 21 years. In all cases the same deformity of crowding of the lateral nasal and lateroposterior epipharyngeal walls of the choana along with atrophy of turbinates was encountered.

The transpalatine approach to the correction of choanal atresia is a very simple procedure that no one with good surgical training should be afraid to perform, and I assure you the results will be most gratifying.

CONCLUSIONS.

1. The exact etiology of choanal atresia is not known.
2. Difficulty in treatment arises because of cicatricial contraction after surgery. This is due to a crowding of the lateral nasal walls and also the lateroposterior wall of the pharynx.
3. A transpalatine approach offers the best means of surgical correction.
4. Surgical correction early with restoration of normal nasal physiology might prevent the poor development of the nasal and pharyngeal cavities on the obstructed side.

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IDIOPATHIC (LETHAL) GRANULOMA OF THE MIDLINE FACIAL TISSUES TREATED WITH CORTISONE: REPORT OF A CASE.*

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Since idiopathic granulomas of the midline facial tissues are being seen with apparently increasing frequency, we think that the following case report is of sufficient interest to justify its presentation. Most cases are not reported either because of the difficulty of making a definite diagnosis, or because in such a long, lingering illness the patient usually goes from physician to physician in hope of relief, so that one physician is not often able to observe the total course of the illness. The outlook appears so hopeless, all patients eventually dying of the disorder, that piling up records of what appears to be an insoluble medical puzzle may seem a futile undertaking. The use of cortisone in this group of patients, however, is encouraging, in that it seems to have a definite effect in producing a healing tendency in the lesions. It is for this reason particularly that we feel that the presentation of this case might be of general interest.

Stewart[1] seems to have furnished the most vivid and accurate clinical description of this disorder. He divided the symptomatology of the disease into three stages. The first stage, or prodromal stage, may last for a month or two, or as long as four to five years. At first the patient may merely be

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conscious of a stuffiness in the nose, sometimes accompanied by a watery or serosanguinous discharge. A superficial ulceration of the septal mucosa, thought to be on a traumatic basis, may appear and disappear. On the other hand, the initial expression of the disease may be a superficial ulcer in the roof of the mouth or in the gingivobuccal groove. Some patients seem to have had infection in the paranasal sinuses preceding the onset of the lethal granuloma; in others a history or findings suggestive of an involvement of these structures cannot be obtained.

The second stage Stewart[1] termed the stage of active disease. The patient's nasal obstruction may be associated with a small, brownish ulcer on the septum, inferior concha or roof of the mouth, covered with a gelatinous crust which on removal shows a shallow ulcerating surface. The discharge is purulent or sanguinopurulent and has a foul smell. The disease tends to spread from the inside of the nose to the palate, or from the inside of the nose to the outside, so that early in the active stage the tip of the nose may become swollen and indurated. A perforation of the hard palate may be one of the early signs. The disease progresses and sequestra of the nasal bones, the malar bones and the palatal bones may come away. Abscess formation may take place under the cheek and may require drainage. Secondary granulomas may appear on the posterior pharyngeal wall, the nasopharynx or the larynx. There may be a little fever with an irregular temperature and very moderate leucocytosis or even leucopenia. The polymorphonuclear leucocytes tend to maintain their normal proportions in the blood picture. Agranulocystosis has never been found and septicemia as indicated by the blood picture is absent. Except for rather pronounced weakness and lassitude, the patient may present a curious impression of well-being in spite of the serious appearance of the advancing lesion.

In the third or terminal stage, Stewart[1] stated that the obvious exhaustion of the patient is an outstanding clinical feature. The facial appearance may be monstrous. The eyelids are swollen and purulent discharge trickles from between

the lids. Sloughing areas appear over the lacrimal sacs and cheeks and the soft tissues about the external nose disappear, perhaps including the upper lip. The mutilating destruction may be extreme. The hard and soft palates may be completely destroyed. Through the large central aperture in the face, the tongue, the roof of the nose and the nasopharynx may be plainly visible, together with the roofs of the maxillary air sinuses exposed by ulcerative destruction of the lateral walls of the nose. The posterior pharyngeal wall may be covered with dirty, necrotic material. The tongue remains unaffected and sequestration of the basisphenoid has never been observed. The patient may die of hemorrhage from ulceration of large vessels which are troublesome to control in a necrotic region, the bony walls of which have been destroyed; however, usually he seems to die from simple exhaustion.

Stewart[1] summarized his clinical description by stating that the picture is one of progressive destruction of nose, face and pharynx. The duration of the illness may be from a few months to five years or more. The most striking feature of the disease is the apparently complete absence of resistance on the part of the patient to the progress of the disease.

Stewart's[1] brilliant clinical description of this disorder can hardly be improved upon.

Weinberg[2] in discussing idiopathic granulomas added what appears to be an important point. He found that these lesions were not strictly limited to the midline tissues of the face, but that here and there throughout the body a pathologic picture of periarteritis nodosa developed.

Teilum[3] stated that, by looking on these lesions from a common pathogenetic point of view, based on morphologic observations and experimental studies on the vascular and tissue changes in allergy, essential support has been obtained for the concept that these lesions are primarily vascular and are of allergic nature.

According to Klinge,[4] the essential morphologic substrate of these related conditions is a swelling of the ground vessels which are involved in the process, with edema, fibrinoid degen-

eration and swelling of the collagen fibrils in all the layers of the walls of the vessels as the first morphologic change. Klinge believed nonspecific granuloma to be an allergic lesion which pathogenetically is closely related to rheumatic fever as well as to allergic vascular lesions such as periarteritis nodosa and lupus erythematosus disseminatus.

Evidence[5] has been presented which indicates that underlying all the physiologic mechanisms of the body for resistance to stress lies a stereotyped reaction of the peripheral vascular bed. This consists of arteriolar constriction with dilatation which may amount to atony of the conjoined capillary and venule. This reaction accompanies antigen-antibody allergy, but it is a more primitive mechanism and may itself produce the clinical manifestations of allergy without the intervention of an antigen-antibody reaction.

Kahn[6] has suggested that allergy is merely an expression of hyperimmunity and that there are many examples in physiology of the overactivity of a physiologic mechanism.

Walsh, Sullivan and Cannon[7] have demonstrated that immunity may be localized to or be greater in certain tissues than others.

The midline tissues of the face is a region where skin and subcutaneous tissues and mucosa and submucosal tissue come in close relationship. These tissues are among those most capable of developing an intense immunity and consequently a hyperimmunity, and consequently it is not unreasonable to suppose that they might readily become the site of a destructive granulomatous reaction of an allergic nature.

Differential Diagnosis: From the purely clinical standpoint it seems apparent that there is nothing in the gross picture of this condition to distinguish it from specific infectious granulomas occurring in the same locations except the appearance elsewhere in the body of lesions of periarteritis nodosa or lupus erythematosus disseminatus or rheumatism.

Pathologists are careful to report sections taken from these lethal granulomas as being not "typical" or "diagnostic" of

this or that condition, being prepared to admit that an atypical example of any one of the specific granulomas might be present.

Hoover[8] gave a typical pathologic description of one of these idiopathic lethal granulomas. He stated, "On the border of the lesion is an amorphous mass of necrosis beneath which there is an infiltration by inflammatory cells, chiefly lymphocytes and macrophages with occasional plasma cells. A few polymorphonuclear leucocytes are also present. The large vessels in the tissue show a definite perivascular cuff of lymphocytes and macrophages with degeneration of arterial walls in some regions. While there are focal accumulations of infiltrating cells in the tissues, the lesion is not characteristic of tuberculosis, syphilis or leprosy." The pathologist added that the lesion suggested the Arthus phenomenon.

Weinberg[2] pointed out that oidiomycosis, glanders, tularemia, leprosy, rhinoscleroma, blastomycosis and sporotrichosis all give a somewhat similar histologic picture and attempts to demonstrate these organisms as etiologic agents should be made. He also considered the possibility that a virus might be the causative agent, but stated that of the viruses, only that of venereal lymphogranuloma produces a microscopic picture which is similar.

Weinberg believed that if these organisms are eliminated insofar as possible, there is left a picture which clinically consists of ulcerations of the upper respiratory tract associated with evidence of focal alterations in the lungs, and which anatomically shows multiple granulomas of unknown origin involving one or more organs with terminal development of periarteritis nodosa. He believed that the constancy of the history of antecedent chronic infection in these cases lends support to the belief that hypersensitivity may be a factor in their development.

The evidence presented seems convincing that idiopathic lethal granuloma of the midline facial tissues is an instance of localized hyperimmunity resulting in an Arthus-like reaction of the involved tissues.

REPORT OF CASE.

The patient is a woman 50 years of age. In November of 1949 the Eustachian Tubes were inflated because of a pounding in her ear. There was some trauma on the right side followed by pain in the nose which gradually grew worse. A week later she observed an ulcer on the right side of the septum. About two weeks later, or around Dec. 1, 1949, she developed a blood-tinged, yellowish discharge from the right nostril associated with burning and tenderness of the nostril. Swelling of the tip of the nose followed, but did not persist. The discharge continued. The process soon extended to the left side and crusting and epistaxis developed. Treatment consisted of the local use of sulfathiazole ointment and oil nose drops and the systemic use of aureomycin, penicillin and one of the sulfonamides.

She was referred to the Mayo Clinic on Jan. 7, 1950. An ulceration in the anterior portion of the septum, extending backward about 2 cm., covered with granulation tissue was found. The rest of the nasal mucosa appeared normal. Sinus X-rays showed a slightly thickened membrane in both antra. There was a secondary anemia, but the differential leucocyte count was normal and blood smears showed nothing of significance.

A biopsy was taken from the nose and sent to the Sections on Pathology and Clinical Bacteriology of the Mayo Clinic. The pathologist reported the tissue to be a granuloma with foreign body giant cells and eosinophiles. There was considerable subepithelial fibrosis. Bacteriologically, the material was studied extensively in various special media, both aerobically and anaerobically. Micrococcus pyogenes and streptococci fecalis were found. Repeated cultures for tuberculosis, Brucella and fungi were negative. They were unable to isolate a virus. There were three negative guinea pig inoculations. Kline, Kahn, Hinton and Wassermann tests for syphilis were negative. A diagnosis of idiopathic lethal granuloma of the midline facial tissues was made.

Comment: Because the evidence from the literature suggested that this condition was an allergy, nonspecific and antiallergic treatment with the vasodilator, monoethanolamino nicotinate (Nicamin Abbott) was given. The patient was dismissed to the care of her home physicians.

The patient seemed to show improvement after first returning home, but in March she developed hoarseness which had grown worse, and this was accompanied by an increasing dyspnea. The ulcerating granuloma was then seen to involve both sides of the septum and the anterior ends of the inferior turbinates. The interior of the nasal cavities could not be inspected because gentle attempts to cleanse the nose of crusts and mucopus produced bleeding. There was a pale granulomatous mass in the nasopharynx. There was granulomatous involvement of the anterior half of the left vocal cord and a smaller granuloma on the right arytenoid. Chest X-rays were negative. She complained of loss of appetite, strength and about 20 pounds of weight. There had been shooting pains in her legs for the previous two weeks. Nicamin was continued and terramycin, 250 mg. every four hours, was added.

From May 5 to June 29 she was on intermittent treatment with terramycin. By May 25 the nasal picture had improved, but the larynx had become more involved and there were large subglottic granulomata. Breathing was embarrassed to the point where a tracheotomy was considered. During the rest of this period, however, the nasal granulomata

became firm and showed less tendency to bleed. There was regression of the subglottic granulomata with improvement in breathing, but none in her voice. This was probably the result of the effect of the terramycin on the secondary infection rather than on the primary process.

On June 28, 1950, cortisone was obtained for her and she entered St. Luke's Hospital, Cleveland, Ohio, for further study and treatment. The patient refused to allow a biopsy from her larynx or repetition of one from her nose. After much persuasion a small biopsy was taken from the nose on July 14 and used for special cultures with the same negative results which had been obtained at the Mayo Clinic.

On admission to the hospital, the following interesting family history was obtained. One sister had leg ulcers with involvement of eyes and lungs which was suspected to be tuberculosis. This is now arrested, but she had many contacts with the patient. One half-brother and a niece had been in a sanitorium with suspected tuberculosis. No definite diagnoses had been established. The patient was found to have papulopustular lesions on the elbows and right foot. Blood chemistry was normal; the glucose tolerance test was normal. She still showed a secondary anemia and there were 10 per cent eosinophiles in the differential count. An intravenous epinephrine test of the pituitary-adrenal mechanism was given. The circulating eosinophile count at 7 A.M. before the epinephrine was 346 cmm. At 10 A.M. this count had fallen to 229 cmm., and at 11 A.M. to 170 cmm., which was considered a normal response.

On July 10 one of the papules was removed from the elbow. This was examined by Dr. Raphael Dominguez and Dr. A. J. Segal. They reported necrosis of the epithelium in the central portion with the necrosis extending beneath intact epithelium on one side. There was a heavy infiltration of polymorphonuclear leucocytes and round cells with no evidence of malignancy. Stains for bacteria and fungi were noncontributory. Their diagnosis was necrotizing granulomatous dermatitis, cause undetermined. Slides of this tissue were sent to the Mayo Clinic where they were examined by Dr. A. C. Broders, who thought that it was a Grade I hemangioendothelioma representing a Kaposi tumor. He reviewed the tissue that had been removed at previous biopsies and agreed that this diagnosis could not be made from these slides. The patient had other lesions on the elbows, thumbs, fingers and feet which were identical in appearance to the one removed from the left elbow for biopsy. On her return to the Mayo Clinic these lesions were observed by a consultant in the Section on Dermatology and it was his opinion that these lesions did not resemble Kaposi's tumor.

The clinical course of this patient, while bearing some slight resemblance to that of Kaposi's tumor, is much more characteristic of that of idiopathic lethal granuloma; moreover, Dr. Frank B. Hazard, of Cleveland, and Dr. Joseph C. Ohlmacher, of Vermillion, S. Dak., pathologists to whom these tissues have been submitted, have not been convinced that the area in question was truly representative of an endothelioma, but have thought it probably representative of the necrotizing vasculitis seen in both specific and nonspecific granulomas. It was, therefore, felt that the weight of the evidence was on the side of the lesion being an idiopathic granuloma and that treatment should be given on this basis. Since idiopathic granuloma was thought to be due to a local hypersensitivity with the stereotyped reaction of the peripheral vascular bed seen in these conditions, the use of cortisone was advised.

Cortisone in Allergic Reactions: Most of the conditions in which cortisone has been found effective have been the disorders which are usually referred to as the "collagen diseases." The evidence suggests[5] that the term "collagen disease" is synonymous with allergy; in other words, localized areas of tissue hypersensitivity. Among the disorders in which the local lesions have responded favorably to cortisone therapy are rheumatoid arthritis, vasomotor rhinitis, asthma, periarteritis nodosa, lupus erythematosus disseminata and certain diseases of the eye, all of which are grouped under the collagen diseases and, therefore, may be reasonably presumed to be allergic. The exact manner in which cortisone influences the functional and histologic lesions in the allergies has not been worked out.[10] Since the evidence suggests that lethal granuloma is also a collagen disease (or an allergy) it was decided that cortisone would be worth a trial in this condition. One of us (Williams[9]) had previously reported a case in which cortisone had apparently had some favorable effect.

The patient accordingly was placed on 200 mg. of cortisone a day for two days and then 100 mg. a day. Four days later she was feeling much stronger and had less general aching. Her breathing was improved and there was less drainage from her nose. The sedimentation rate was 41 mm. per hour. The tenderness of the skin and joint lesions had disappeared.

We were concerned over the possible effect of cortisone on the healing of the site of the biopsy. This, however, healed normally and the sutures were removed on the fourth day.

Nine days after starting cortisone the glucose tolerance was decreased and she was put on a diabetic diet and iron. The sedimentation test was approaching normal and the circulating eosinophile count was within normal limits.

On July 21 she was discharged from the hospital on 50 mg. of cortisone a day, ferrous sulphate and potassium iodide. At that time the turbinates appeared atrophic and there was a great deal of nasal crusting. The vocal cords were still red, but the granuloma on the left cord was barely visible. A week later there was a definite depression of the bridge of the nose

and a large perforation could be seen in the septum. The granuloma appeared to be markedly decreased and dense scar tissue was apparent in the area. The voice gradually increased in strength and her general strength and weight improved. This patient, however, remained on 50 mg. daily for 41 days and while there was no further improvement in her condition, neither did it regress, the *status quo* being maintained.

Cortisone was stopped on September 2, and three days later pain and swelling of both ankles returned. She returned to the Mayo Clinic and by Sept. 11 she was having severe dyspnea from an increase in the subglottic granulomata. She was seen by a consultant in the Section on Rheumatology and a diagnosis of rheumatoid (atrophic) arthritis of the knees and ankles was made.

There had been a very marked change in the lesion in the anterior portion of the nose since she was last seen. The granuloma in this area had entirely disappeared and was replaced by dense fibrous tissue which had narrowed and deformed both nasal vestibules. The bridge of the nose in the area between the upper and lower lateral cartilages was pulled in. A large septal perforation was present. The area in the nose resembled the scarring of rhinoscleroma. There was a granulomatous mass in the nasopharynx.

Inspection of the larynx showed diffuse involvement of both vocal cords, but the major involvement was in the subglottic region. A tomogram taken of the laryngeal region was reported to reveal a large, soft tissue mass in the region of the thyroid and cricoid cartilages which was producing lateral compression from both sides in the subglottic region. The patient was seen by a consultant in the Section on Laryngology who advised emergency tracheotomy. Because of the nature of the involvement and the previous good response to cortisone, it was considered wiser again to try the effect of this hormone.

The skin lesion on the right foot had also enlarged and ulcerated. The dermatologic consultant thought that this

lesion might represent a necrotic dermatitis of the type seen in lupus erythematosus disseminatus.

The Cortisone Committee of the Mayo Clinic allotted enough cortisone for three weeks' treatment.

The patient was placed on 200 mg. of cortisone daily for four days. This was followed by 100 mg. a day until the end of the three weeks' treatment period, Sept. 26. The pain and stiffness of the knees and ankles had entirely receded by the end of the third day of treatment, the ulceration on the right foot had nearly disappeared, the patient's dyspnea was entirely relieved, and her voice was more normal. Another tomogram of the laryngeal region taken at this time was reported to show a 50 per cent reduction in the size of the soft tissue mass with disappearance of the encroachment on the airway.

The patient was again dismissed to the care of her home physicians; but three days later, due to a recurrence of the arthritic pains in the knees and ankles, the cortisone had to be resumed. It was again stopped on Nov. 25.

During the first two weeks of December the patient had abdominal pains, diagnosed as being due to a pancreatitis with calcification. She also had some blood in her urine, and tests showed the kidney function to be decreased. Except for these episodes she continued to improve and 18 days after she had discontinued cortisone the vocal cords looked relatively normal and the subglottic granulomata had almost disappeared; however, at the end of four weeks she again became dyspneic and the subglottic granuloma was large and red. She was again given cortisone.

Comment: By Dec. 28 she had received about 9.5 gm. of cortisone. Except for its effect on her glucose tolerance and some possible kidney involvement, she has shown no ill effects from its continued use. When the cortisone has been stopped, the granulomata have increased in size rather promptly, but the last period before relapse was about four weeks as against only a few days previously; however, at no time has she been completely free of her lesions except for the vocal cords and the skin.

During the course of her illness she has shown granulomata of the nose, nasopharynx, larynx, trachea and skin of the elbows, thumbs, fingers and feet. She has also shown some episclerotic injection of the eyes. At one time while at home she had a red, painful swelling of the left auricle which subsided without further involvement. We feel that the most encouraging feature in this case is the replacement of the initial lesion of the disease in the anterior portion of the nasal cavities by scar tissue. We hope that continued use of cortisone will encourage such changes in the granulomatous lesions in the nasopharynx and larynx.

Summary and Conclusions: A 50-year-old woman developed an idiopathic lethal granuloma of the nose which secondarily involved the nasopharyngeal and laryngeal regions. A rheumatoid arthritis of the knees and ankles developed synchronously with the laryngeal involvement, and lesions resembling the skin lesions of lupus erythematosus disseminatus developed on the elbows, hands and feet.

The results of cortisone therapy appear to encourage further trial of this hormone in this previously lethal disease.

At this time (March 15, 1951) there is no visible granuloma in the nose, nasopharynx, larynx or trachea. The skin lesions have disappeared. This has been the case since Feb. 20, 1951. On Feb. 18, 1951, she was switched to ACTH, 25 mg. every six hours. This was reduced on Feb. 21, 1951, to 10 mg. four times daily. She still has some abdominal discomfort, nausea, blood and albumin in the urine and generalized edema. Since being put on ACTH she has developed marked pigmentation of the skin typical of that seen in Addison's disease. There is no pigmentation of the mucous membranes.

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INTRALARYNGEAL ARYTENOIDECTOMY WITH OBSERVATIONS IN THREE CASES.*

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I appreciate this opportunity of appearing on the program, and I should like to speak on the subject of intralaryngeal arytenoidectomy and present some of my observations concerning three cases.

Case 1: Forty-five-year-old Mrs. E. D. became known to us as "Calamity Jane." An an adopted child without any knowledge of her family, she did very well until the age of 19 when she had an appendectomy, and 19 years later she had a kidney operation which was followed by partial right hemiplegia from which she recovered. The next year brought an operation for adhesions, and three years later there was incised and drained a rectal abscess. This brings us up to 1946. Shortly after the rectal abscess, she developed signs and symptoms of thyrotoxicosis and enlargement of the thyroid gland. On Feb. 23, 1947, my examination of her larynx gave essentially normal results in all respects. I did not see her again until April, 1947, but the story goes that on Feb. 24, 1947, she was operated upon for thyroidectomy and during the course of the operation she developed dyspnea, for which a tracheotomy was necessary. Microscopically, tissue from the thyroid gland proved to be malignant, and during the weeks following the operation the malignant tissue grew out into her neck and exuded through the thyroid incision. Ninety-nine hundred Roentgens of X-ray therapy produced astoundingly gratifying results. On April 16, 1947, I saw this patient again, and at that time there was complete paralysis of the right vocal cord; but there was a little movement of the left vocal cord. She apparently had adequate breathing space, the tracheal tube was removed on April 20, 1947, and she was allowed to go home. Her voice was poor.

Seven months later, using local anesthesia, a tumor was removed from one arm, and this proved to be benign. During the course of the next year she developed pain in both arms and in December, 1948, under pentothal sodium anesthesia, cervical laminectomy was performed for relief of the pain. During the course of the operation, she developed acute dyspnea, and a tracheal tube had to be inserted. This tube was removed about two months after the operation.

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During the months that followed this last operation, she noticed that her throat had become more sensitive, and that she had noisy inspiration. There were attacks of laryngeal spasm. Her friends told her that she snored loudly at night. She was examined in my office some months after the last operation, and being unaware of her recently developed sensitive throat, I inadvertently set off an attack of laryngeal spasm by placing a laryngeal mirror in the oropharynx. The spasm lasted for about 15 minutes, and caused cyanosis and considerable fright to the patient. Attempted examination in the hospital under mild sedation was followed by another attack of laryngeal spasm which was of longer duration than the office attack and necessitated the administration of oxygen. Because of the increasing dyspnea and the attacks of laryngeal spasm, a size 5 tracheal tube was reinserted with complete respiratory relief. Obviously she had not had adequate airway. By laryngeal examination it could be seen that the right vocal cord remained immobile, and there was the same slight movement of the left vocal cord.

On Nov. 7, 1949, she was operated upon for arytenoidectomy by the method to be described. Two weeks later the tracheal tube was removed, and in approximately four weeks from the time of operation she was dismissed from the hospital.

Since the arytenoidectomy, she has not had respiratory difficulty, and there has not been an attack of laryngeal spasm, even when the larynx has been indirectly examined; moreover, she has not had any operation during these 14 months. Her voice remains much the same as it has been since the original thyroid operation.

Case 2 is that of 55-year-old Mrs. A. G., who was operated upon for thyroidectomy on Sept. 27, 1949. She was unable to talk when she awoke from the anesthesia, but there was no dyspnea. For three months her voice improved, but then she began to have occasional "strange, loud and heavy" noises to erupt spontaneously from her throat, and there was shortness of breath on exertion. Six months after the operation she suffered a severe attack of dyspnea, which was followed in three days by a second such attack, because of which a tracheotomy was done by her general surgeon and the anesthetist. Mirror examination showed that she had bilateral abductor paralysis of the vocal cords. Right arytenoidectomy by the method to be described was performed one week after the tracheotomy. Her tube was stoppered in two weeks and removed in four. By mirror examination, it seemed that she had an excellent result, and she felt that she had adequate breathing space. Her voice was good. Seven months later, however, she came to my office for an interval visit, and there she stated that she was breathing and talking all right, but she was so tired all the time that she felt that she could not live. She was most tired when she awoke in the mornings. Inquiry of her neighbors brought out the fact, completely unknown to her, that her breathing at night was so loud that it disturbed them. Those persons living in apartments closed to the patient were very desirous of moving elsewhere. Three days later I reinserted the tracheal tube, and on the following day she told me that she awoke from the anesthesia "rested." She went back to work in a few days, and she said that she felt so well that on her first day back in the office she caught up on the work that had accumulated during her absence. Obviously her operation for arytenoidectomy to relieve respiratory obstruction due to bilateral abductor paralysis thus far has failed. Further operation is contemplated.

Case 3 is that of a 38-year-old colored farmer. His story began with a gunshot wound of the throat while he was in the army in 1942. From

this he recovered. In 1946, he was operated upon for thyroidectomy. On the same day of the operation there was acute dyspnea, and tracheotomy was performed. Three months later the tube was removed. Breathing was described as "fair"; voice "poor." For four years his condition was written off as "fairly satisfactory," but there were occasional mild attacks of shortness of breath. Then in July, 1950, four years after the removal of the tracheal tube, he developed severe shortness of breath, requiring emergency tracheotomy. Examination revealed that he had bilateral abductor paralysis of the vocal cords. Within a month right arytenoidectomy was carried out by the method to be described. Two weeks postoperatively the tracheal tube was removed, and he was dismissed from the hospital 10 days later. At the time of his discharge from the hospital he had very adequate breathing space, and his voice was fairly satisfactory. His course since that time is unknown since he does not communicate with us.

A study of the surgical anatomy involved in the operation for intralaryngeal arytenoidectomy was previously done in the laboratory.

Technically, the actual operation is done in a similar fashion. Since these patients come to the operating room wearing tracheal tubes, the maintenance of adequate airway is not a problem. One and one-half hours before the operation nembutal, gr. 1½ is administered by mouth, and one hour before the operation morphine gr. 1/6 together with atropine gr. 1/150 is administered by hypodermic injection. When the patient is brought to the operating room, the nose and throat are sprayed with a 4 per cent solution of cocaine hydrochloride. The patient is then generally anesthetized with pentothal sodium intravenous anesthesia, and the larynx is sprayed with a 10 per cent solution of cocaine hydrochloride through a Jackson laryngoscope. The Lynch suspension apparatus is inserted. In the event that the patient's upper front teeth are precarious, an acrylic mould is previously made. This fits over the front teeth and is long enough anteriorly so that it rests against the anterior nasal spine. Pressure of the suspension apparatus against this mould is transferred to the nasal spine and assists in taking the pressure off the upper front teeth. When the suspension apparatus is inserted, it should be remembered that only the posterior part of the larynx is to be exposed, and this may be accomplished without elevating the epiglottis. This is helpful in preventing edema

of the epiglottis which seems to delay subsidence of the operative edema of the arytenoid region.

The operative setup involves only some seven instruments: (see Fig. 1) a long knife handle which carries an 11 or 15-

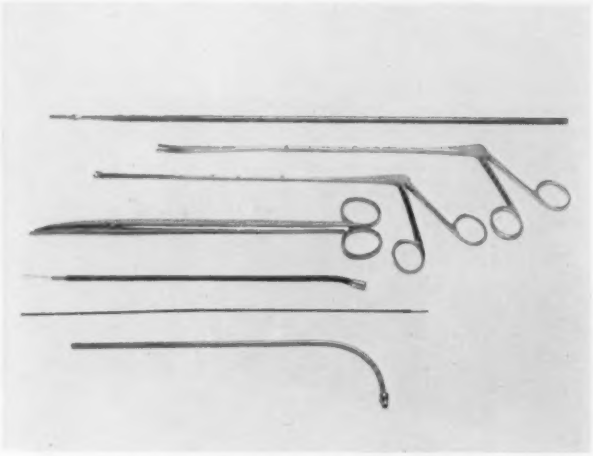

Fig. 1. Instruments used in the operation.

blade, two laryngeal grasping forceps, a pair of long, slender scissors, a laryngeal electrode, a laryngeal needle and a laryngeal suction tip.

With the larynx exposed (see Fig. 2), the mucous membrane over the condemned arytenoid cartilage is injected with a solution of 1.5 per cent monocaine hydrochloride in which there is epinephrine 1:100,000, through the 22-gauge needle of 11-inch length. This increases the depth of anesthesia locally, and greatly aids in hemostasis.

Using an 11 or 15-knife blade on the laryngeal length knife handle, an incision is made along the medial two-thirds of the aryepiglottic fold. The incision is carried down to the corniculate cartilage. Then by the use of long scissors, the mucous

membrane is dissected from the arytenoid cartilage. As soon as convenient (see Fig. 3) the arytenoid cartilage is grasped with laryngeal alligator forceps, and the dissection is completed with scissors and knife. After the cartilage is removed

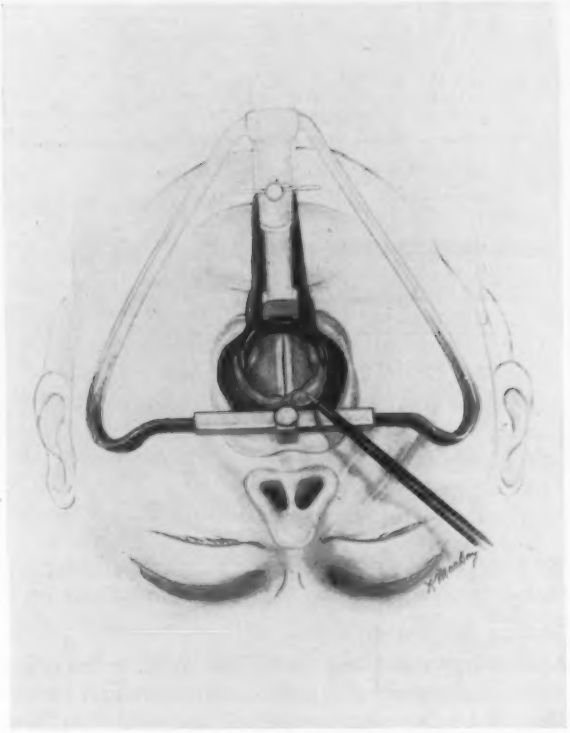

Fig. 2. Exposure of the larynx by means of the Lynch suspension apparatus. Mucous membrane over the condemned arytenoid cartilage is injected with monocaine hydrochloride solution.

hemostasis is accomplished by the use of the electrocoagulation current through the laryngeal length electrode. Finally (see Fig. 4), a mattress suture of 00 chromatic catgut is placed so that it enters the lateral aspect of the aryepiglottic

fold, passes through the posterior end of the vocal cord, and is passed back through the aryepiglottic fold close to its point of entry. It is not the purpose of this suture to close the incision; it serves only to pull the cord laterally. The incision is not sutured. This provides adequate drainage for serum and blood.

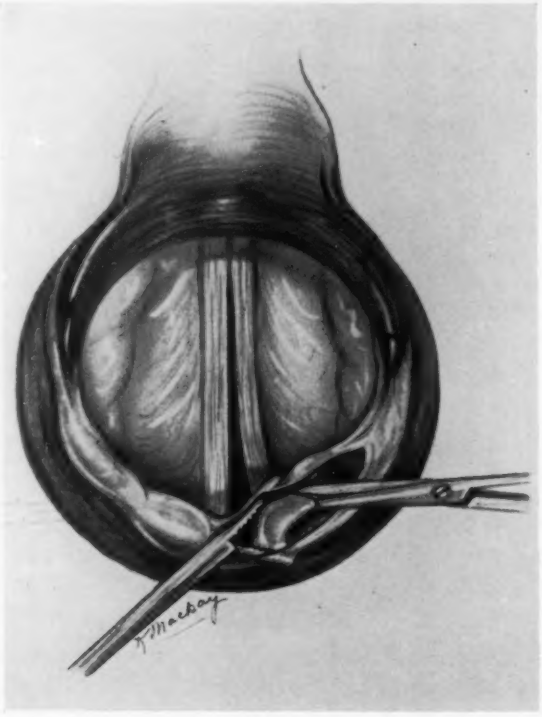

Fig. 3. Removal of the arytenoid cartilage.

Postoperatively for the next three days the diet is liquid, partly because the patient's throat is sore, and partly because it facilitates a cleaner throat. The patient may be up as soon as the effects of the anesthesia have subsided. The tracheal

tube is stoppered at about 10 days, and removed in two to three weeks, according to the results of the mirror examination, and the patient's symptoms.

There are several advantages to this method: *a.* Simplicity. The operation is less formidable, as a rule it is less time consuming, and the arytenoid cartilage is so much more easily located. *b.* The possibility of wound infection is reduced. *c.* There is less likelihood of the postoperative formation of hematoma which may cause a reduction in the calculated

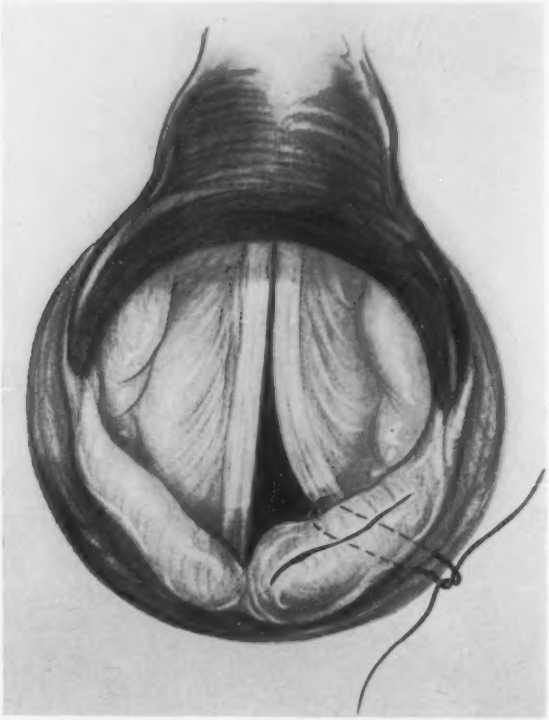

Fig. 4. Placement of the mattress suture to laterally displace the vocal cord.

width of the glottis. *d.* When working directly in the larynx, the surgeon has immediate and constant view of the amount of space that is being created between the vocal cords.

There are only two disadvantages: *a.* Rarely there may be technical difficulties in the use of the Lynch suspension apparatus. *b.* There arises the question of whether the vocal cord can be displaced far enough laterally without suturing it to the wing of the thyroid cartilage. Only statistics will answer.

As to when the patient should be operated upon for bilateral abductor paralysis of the larynx, the consensus of opinion is that if the paralysis remains total six months postoperatively it can be considered permanent.

For the results in these three cases let us consider that there are three classifications of results: the *good* without reservation, the *good* with reservation, and the *poor*. By the second classification I refer to those cases which fulfill some of the criteria for a successful result, but do not fulfill all of the criteria. They may be able to get along, but they have not been returned to the preoperative calculated rehabilitated state. Classes 1 and 3 are self explanatory.

From this classification there might be some individual opinion as to the results. At worst, the batting average would be 33 per cent and at best, 66 per cent. I rather incline toward the latter, since in my opinion, the patient described in Case 1 has been rehabilitated, and so far as we know, the patient in Case 3 has been rehabilitated. Case 2, at the time of this writing, provides us a failure.

I should like to emphasize that in considering the treatment of bilateral abductor paralysis of the larynx, it is necessary to consider many things: the size and shape of the larynx, the sex of the individual, the patient's psychology. The result that could be called a success for the person who does sedentary work might be called a failure for the laborer. The result that could be called a success for the person who does not have to use his voice except to make his wants known might be a failure in a clergyman or a teacher. If time should prove

that similar results can be obtained by the extralaryngeal and the intralaryngeal approaches, the latter will surely have one thing in its favor: ease and simplicity of procedure.

In conclusion, I pay my respects to some of the workers in the field of the treatment of the symptoms of bilateral abductor paralysis of the larynx, Hoover,[1] Loré,[2] King,[3] Kelly,[4] and two members of our present company, Dr. Clerf[5] and Dr. Jackson,[6] but to William C. Thornell,[7,8] of Cincinnati, duly goes the credit for first having brought to the surgeons' attention the intralaryngeal approach to arytenoidectomy.

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137 Doctors Building.

OSTEOMATA OF THE FRONTAL SINUS WITH SPECIAL CONSIDERATION OF THE SURGICAL REMOVAL.*†

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When the literature regarding this essentially or histologically benign group of tumors is reviewed, one is surprised at the various moot questions that are presented. Among them are: *1.* pathogenesis; *2.* base of origin or attachment; *3.* frequency of incidence; *4.* therapeutic indications; and *5.* possibly, the surgical procedures and technique to be followed.

Pathogenesis: Carmody[1] makes clear the controversial opinions, as to etiology, that have been presented, naming four theories:

1. That they arise from embryonic cartilaginous cells at the junction of the ethmoidal and frontal bones.[2] (Arnold.)

2. That the origin is from the periosteum of the frontal sinus walls.[2,3] (Fettisoff.)

3. That the diploe is the generating base.[2] (Virchow.)

4. That some inflammatory stimulus is the underlying reason, suggesting the possibility of lues, tuberculosis, trauma,[2] (Cushing) or sinusitis.[4] (Eagleton.)

5. Another is added by Cloquet[2] who believed them to be ossified polyps.

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On the other hand, Leeds[5] believes that no completely satisfactory explanation has yet been submitted.

Kelemen,[6] in one of his four reported cases, saw histologically an area that reminded him of otosclerotic bone changes.

He also believes that, as a rule, the symptoms are expected to disappear after proper radical surgery.

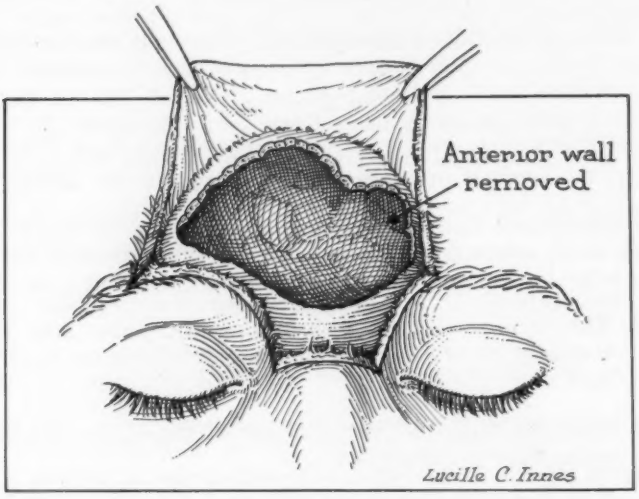

Fig. 1. Case 1. M. J. Removal of anterior plate by gouge and rongeur. There was more bone removed than shown, including some of the left anterior plate and the right supraorbital ridge, making for postoperative deformity.

Austin J. Smith[7] feels that there is a question as to whether the osteomata are true tumors. He states that pathologically (histologically) they are related to the so-called fibrocystic group of bone lesions.

H. Brunner and Spiesman[8] point out that there are two types of expansion of osteomata: some expand by increase in the size of the tumor; others expand by the formation of mucoceles. (Noted within the orbit in this current Case 1.)

Fig. 2. Case 1. M. J. Photograph taken a few years after surgery, showing the marked depression.

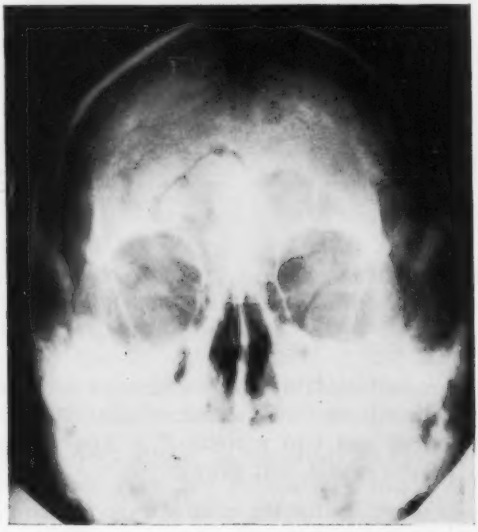

Fig. 3. Case 2. E. J. Preoperative X-ray film showing large tumor.

The increase in the tumor size is due to a metaplastic ossification of the connective tissue and to a deposition of new bone by the paranasal mucosa which serves as periosteum. The tumors thus consist of a spongiose core, and a solid cortex.

Osteomata of the frontal sinus are divided into two classes histologically:[9]

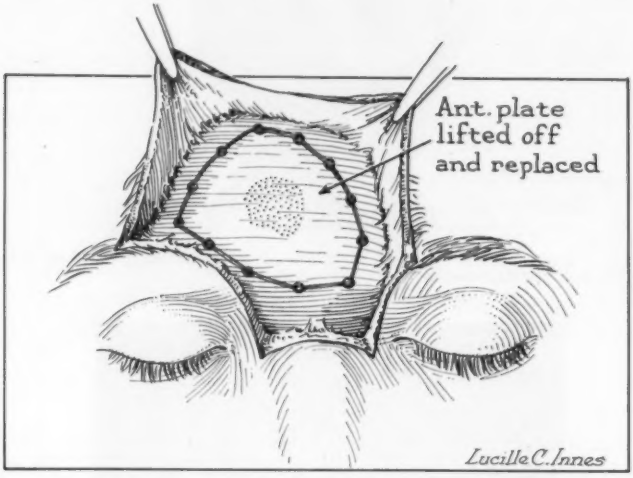

Fig. 4. Case 2. E. J. Lid operation, showing removal and later replacement of the area of anterior plate. The dotted area indicates the bone erosion on the inner surface caused by pressure of the tumor.

1. Hard, dense bone; and *2.* relatively soft, spongy, cancellous bone. There is another classification into two varieties:

1. Pedunculated, and *2.* sessile.

Study of a considerable number of case histories leads one to wonder whether these two methods of classification are not the description of just two varieties: *1.* The pedunculated, hard tumor; *2.* the sessile, soft growth.

Further consideration of the clinical reports brings one to the conclusion that, probably the pedunculated hard tumor is

indeed a true neoplasm, springing from a soft generative base, quite likely, and most usually, an embryonal rest. On the other hand, the sessile, soft mass is often described as accompanied or preceded by evident widespread bone changes. Practically all of the frontal area, or even more or less of the

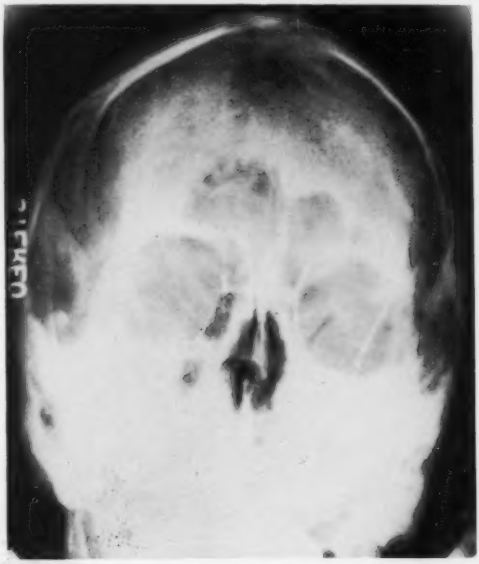

Fig. 5. Case 2. E. J. Postoperative X-ray film; after five months.

skull in general, may reveal X-ray evidence of thickening and softening. It may be that the real etiology is a metabolic bone hypertrophy and that the so-called osteoma in the frontal sinus is only the unbridled thickening, invited by the empty chamber; and in fact is not a neoplasm but a local manifestation of hyperostosis, due to metabolic disorder.

When the hard tumor is loosened from its bed, the soft generative bone may and should be easily curretted away until the underlying normal appearing bone *is reached.* When the soft, spongiose growth is surgically attacked, underlying

normal appearing bone is *not* reached. If the dissection of the soft bone is followed it leads to the end of all osseous tissue, and one arrives within the cranial cavity, with dural exposure. This may explain the advice of some operators that it is not necessary to remove all of the growth and that the

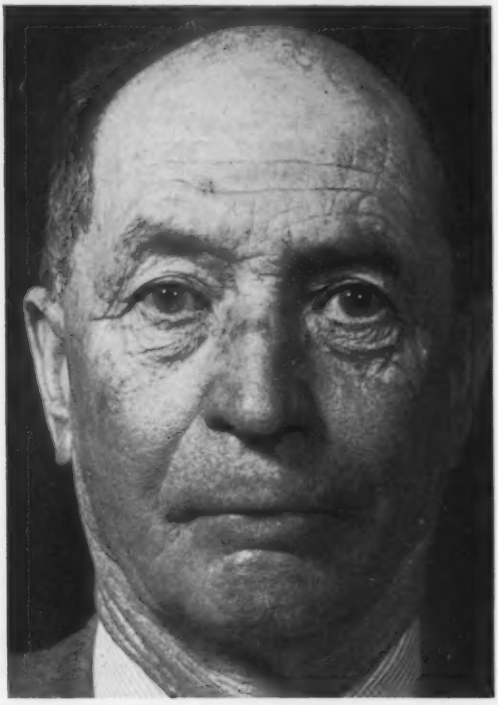

Fig. 6. Case 2. E. J. Postoperative photograph, showing dimple or retracted scar where the area of bone erosion was. Picture taken nearly six months after surgery.

partial removal is not followed by recurrence. Coates describes carefully approaching the depth of the dissected excavation so as to leave a tissue paper thin layer of this bone over the dura, avoiding exposure. When, as in his case, the

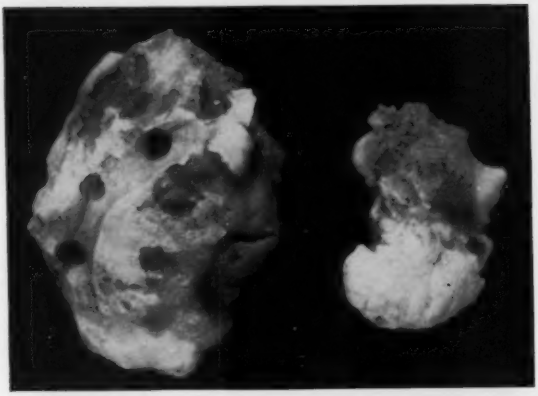

Fig. 7. Case 2. E. J. Osteoma removed in two pieces. Unfortunately, a scale was not placed beside the bone masses. The unbroken mass was approximately 33 mm. broad by 16 mm. deep, and 31 mm. from bottom to top.

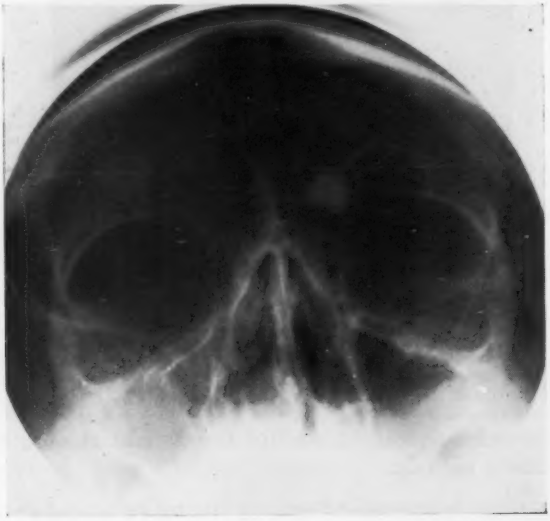

Fig. 8. Case 3. C. B. X-ray film 12 years preoperative.

dura is actually exposed, he advises leaving bone chips over the dura to encourage re-formation of bone.

The Base of Origin: Obviously the variations in the possibilities of base run with the numerous theories of etiology, and many are to be found in the literature.[1,10]

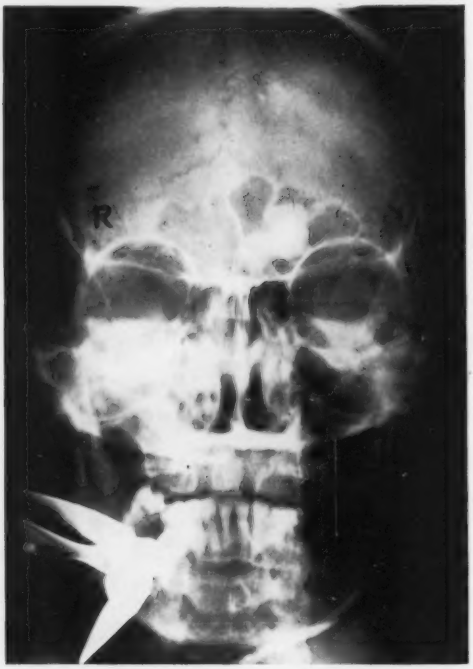

Fig. 9. Case 3. C. B. X-ray film shortly preoperative, showing marked growth of tumor in the 12 years.

1. Junction of the frontal and ethmoid bones, far forward.
2. The under and inner wall of the frontal bone and the ethmoid capsule.
3. A pedicle inseparable from its frontal bone and the ethmoid origin.

4. Continuous with the frontal bone at the inner wall of the sinus.

5. The upper lateral wall of the frontal sinus.

6. The infundibulum.

7. The floor of the frontal sinus external to the duct.

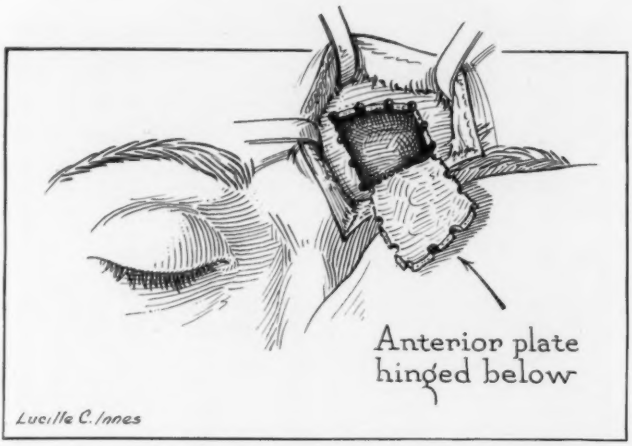

Fig. 10. Case 3. C. B. Hinge operation, showing technique of loosening the anterior plate area on the lateral and superior borders, with the bony area then swung downward over the eye, preserving the soft tissue hinge and thus the blood supply from below.

8. The anterior end of the junction of the ethmoid with the frontal bone.

9. The septum between the frontal sinuses.

Sprowl[2] states that they are usually attached by a cancellous pedicle to the base. In surgical removal, he declares that not only the tumor mass and its pedicle but also the base must be removed to prevent recurrence.

Frequency: The position taken on this question by various observers naturally is largely influenced by their own clinical

experience. Certainly the literature does not list any great number of cases, studied and reported.

Dr. Gilbert Owen, who is to discuss this paper, from the standpoint of his observations of referred cases studied by

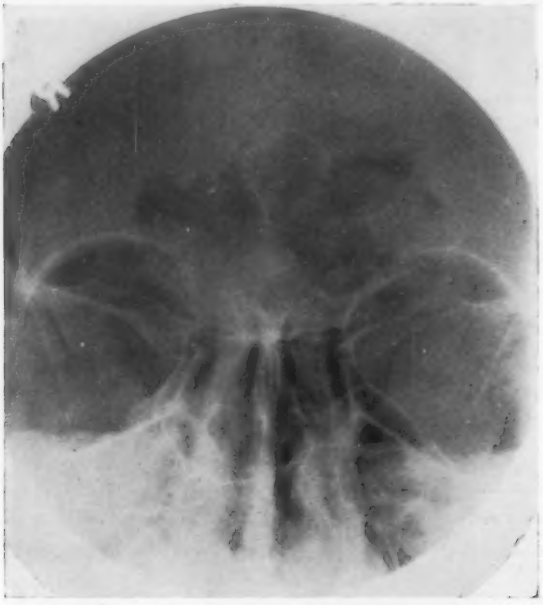

Fig. 11. Case 3. C. B. Postoperative X-ray film showing normal bone texture.

the X-ray, might, reasonably take the position that these tumors are not infrequent. He has seen a series of over 30 cases.

On the contrary, the author found his first case only after 17 years in the specialty. At that time he had never been fortunate enough to have seen a single case operated upon or diagnosed in any clinic, conference, or postgraduate course, here or abroad. His second case did not appear until 22 years

later. This experience would tend to lead to the conclusion that such tumors are quite infrequent, if not, indeed, rare.

Carmody,[10] in 1935, collected 139 cases, giving references to the reporting articles, with the author and the journal.

In a series of 3,510 Roentgenograms of the nasal sinuses,[4] osteomata were noted in 0.42 per cent. This seems to have

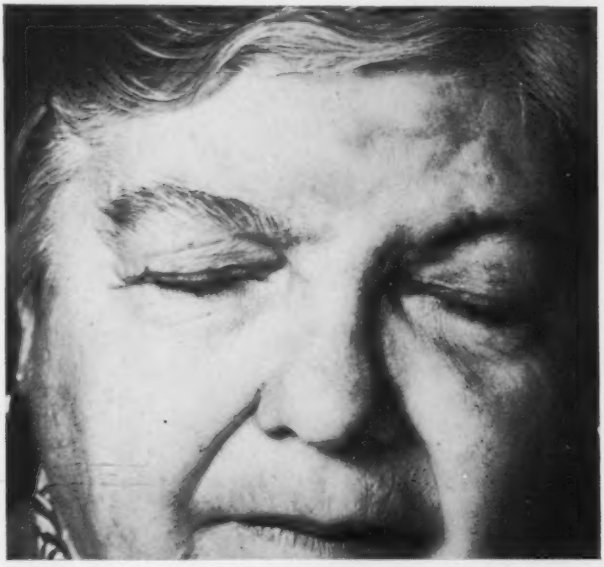

Fig. 12. Case 3. C. B. Postoperative appearance of forehead. As the days followed, there was no untoward reaction or swelling.

been a rather surprising observation. The first osteoma to have been found by Roentgen examination was reported by Cappy[5] in 1899.

In 1935, Beck stated that in his practice he had had four cases.[10]

In 1936, Sprowl[2] took the position that osteomata of the

nasal accessory sinuses were uncommon, the greatest number having been observed in the frontal sinuses.

Reeve,[11] as early as 1918, joins Friedenberg in offering an apology for reporting two cases of his own (one seen in 1881 and the second in 1895) because "of the number of cases already on record." His own excuse was, "That it is desirable to make statistics as correct as possible." Nevertheless, in discussing Reeve's paper, Edward Jackson calls such a tumor

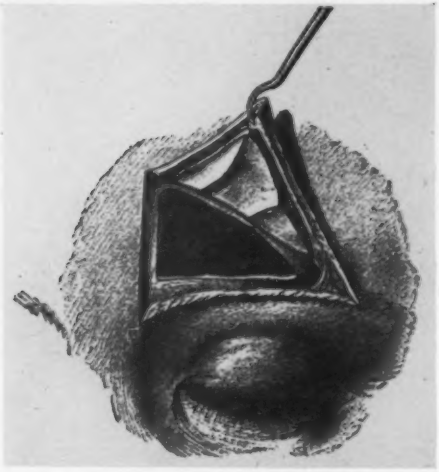

Fig. 13. Showing position of the skin-bone flap in osteoplastic resection of the anterior wall of the frontal sinus. (Illustration taken from Prof. M. Hajek's book, Pathology and Treatment of Inflammatory Diseases of the Nasal Accessory Sinuses, published by C. V. Mosby Co., St. Louis, Mo., 1926, and translated into English by Dr. J. D. Heitger and Dr. French K. Hansel.

a "rare condition." By that time he had had only one case in his own practice, and that some 26 years previous. He had seen two other cases in consultation.

In that same year, Sewell[12] presented a most interesting article. He considers several of the moot questions discussed in the current paper. He mentions that in 1880, Berlin advised the enucleation of an eye, rather than to try to remove

a tumor that had broken through into the orbit. He quotes William MacKenzie as stating, in 1854, that no case of frontal sinus osteoma had appeared in the literature. In discussing the origin as taking place in cartilaginous rests, he does not refute the possibility on occasion, but affirms that this explanation alone cannot be accepted for all such tumors. In his paper, in 1918, he reports that his case was the ninety-third

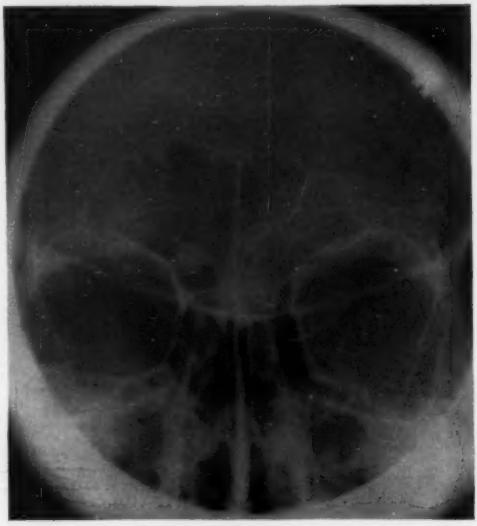

Fig. 14. Case 4. M. S. Preoperative X-ray film showing small osteoma in a large sinus but with clinical symptoms.

to appear in print to that date. Among the cases, in the pathology, under the etiology he does not discuss the sessile type of tumor. He is most enthusiastic in regard to the Roentgen rays being the chief means of making a diagnosis.

In 1938, Malan collected 178 cases of osteoma of the frontal sinus.[13] In 1941, Leed[5] found 321 cases, a figure which he felt indicated that osteoma, while not common, is not rare.

Treatment: These neoplasms are benign and without gen-

eral serological or cytological changes. They occur and develop, as a rule, within a hollow chamber that accommodates them, usually for a matter of years, with no revealing symptoms. In due course, the slowly enlarging mass impinges on one or the other of the frontal sinus walls, so slowly, so gently, that resulting symptoms are not explosive or disruptive to the peace of mind of the victim. Obviously, with all

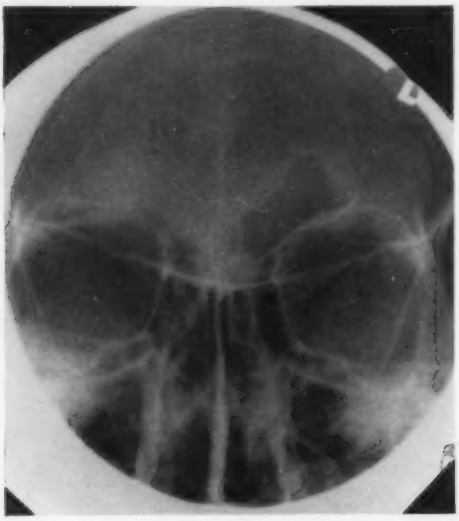

Fig. 15. Case 4. M. S. Postoperative X-ray film showing osteoma gone and with the clinical symptoms relieved.

the nearby vulnerable structures, definite symptomatology nearly always eventually occurs; in fact, the tendency is to malign encroachment on some of the neighborhood tissues[14] by erosion of the inner wall, the anterior wall, the orbital wall, or frequently, by interference with normal drainage.

Hart,[12] in 1943, reported a case that apparently sprang from the right ethmoid area and, before being studied by him, had extended to occupy a large part of both frontal sinuses.

In spite of this most extensive growth, the posterior walls of the frontal sinuses were (Roentgenologically) apparently intact; however, in the ensuing surgery one small area of bone erosion and dural exposure was found. Recovery was uneventful.

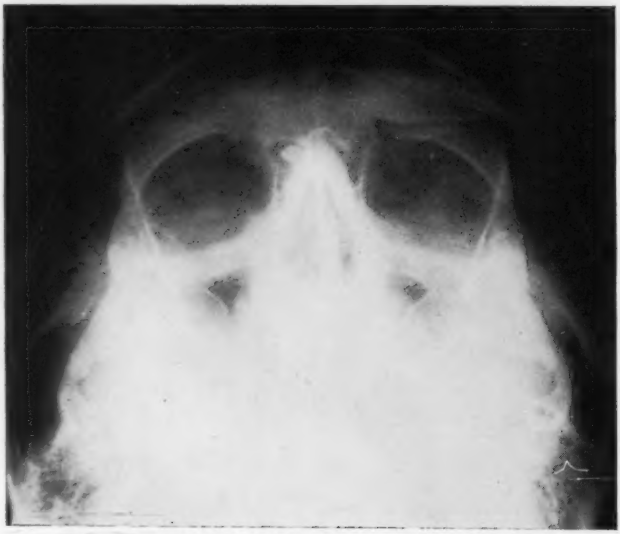

Fig. 16. Case 5. E. H. X-ray film showing a very small osteoma in a very small sinus. The tumor is silent so far.

Hart also quotes Gatewood and Settel as saying that up to 1898, only 20 cases had been attacked surgically, with a mortality of 45 per cent.

Begley and Hollberg[15] advise conservatism in considering surgery. They agree that with definite signs and symptoms present such treatment is positively indicated. Coates and Krauss[9] take the position that, being entirely surgical, as soon as there are enough symptoms to warrant a diagnosis the treatment should be immediate removal.

The chief symptoms are: external deformity; headache or pain, cerebral paralysis; nasal discharge; vertigo; proptosis; and diplopia. W. H. Johnston[16] reported a case in 1943, where the most prominent symptom was dizziness, the next being headache.

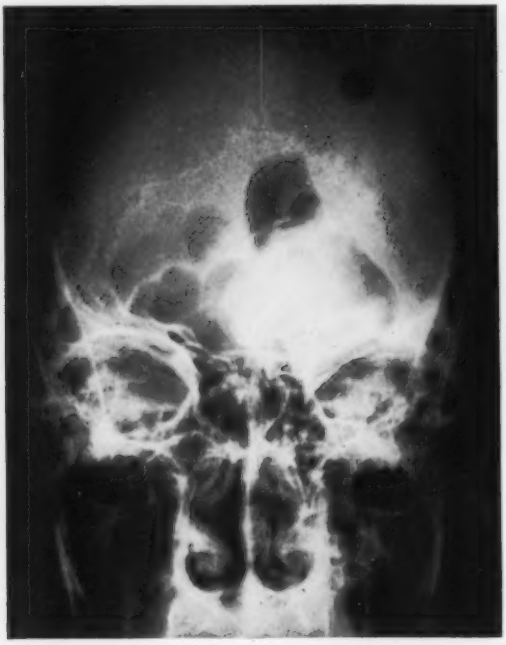

Fig. 17. Case 6. A. R. A-P X-ray film showing enormous bony tumor and peculiar shadows and apparent vacuum or cavity in the frontal cerebral lobe.

It is evident that usually when the tumor is relatively small in a capacious sinus, there is no justification for urging immediate surgery; however, the patient should be instructed as to the possibilities, and have an examination at least every two or three years. Case 3 establishes what may happen in the course of a dozen years. Case 5 shows that size of the tumor alone is not the criterion, as a very small tumor in a very

small sinus may demand early interference. Certainly Cases 1 and 2 demonstrate signs and symptoms that positively necessitate no delay in surgical removal. Case 4 reveals a

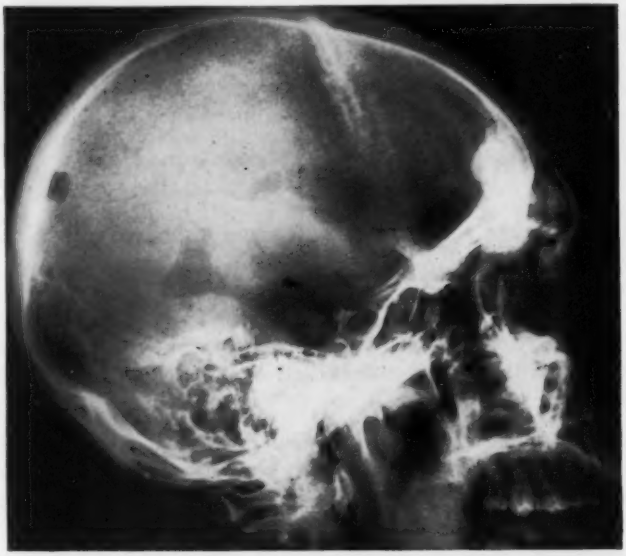

Fig. 18. Case 6. A. R. Lateral X-ray film showing tumor mass in frontal sinus, thickening or opacity in area of posterior plate, and peculiar abnormal shades and shadows in the frontal cerebral lobe.

tumor evidently relatively small, with no apparent impingement, but with clinical symptoms which were relieved by surgery.

Because of continuous, even though slow, growth, encroachment, erosion, pressure and encouragement of extension or invasion of infection, eventually bring on various symptoms, the possible extension and the resulting destruction it will bring is unpredictable. Secondary infection in unoperated cases has been the chief cause of high mortality in the past.[12]

Boher[17] points out that pneumoencephalus is likely to result whenever an air filled sinus, such as any of the paranasal

sinuses, has an interruption in its continuity and air enters the subdural space or the subarachnoid space. In some cases the air may penetrate into the lumen of the ventricular system. He noted vomiting and papilledema as significant symptoms.

Case 6 makes clear the very serious complicating possibilities of neglect or overlooking of symptoms that should warn and indicate much earlier study and necessary surgical action.

Varying and doubtless in many cases quite long periods of silence are followed by clear-cut signs and symptoms. With the increase of X-ray study of sinuses, detection of these silent and unsuspected tumors has probably shown an equivalent increase. These studies have also increased the number of successful results in handling such growths.

In the years before antisepsis[10] the surgical mortality was 31.2 per cent, while since 1875 it has fallen to only 3.7 per cent.[5]

Surgical Procedures and Technique: In reviewing the literature of the past 20 or 30 years, when surgery is indicated, the various authors so state and proceed to remove surgically, without much discussion as to method or technique. It is probable that the usual surgery of the frontal sinus exemplified by the Killian, Lynch or Lothrop techniques was utilized, or the orbital approach mentioned by Benedict and Beck in a discussion in 1931.[8]

Pilcher,[19] in 1938, recorded a successful operation on an osteoma of the frontal sinus by the transcranial approach; however, in contrast to the general clinical reports, Vadala and Somers[20] made a thorough study of the surgical approach and technique.

1. A horseshoe temporal flap (Cushing) with a transcranial approach.

2. A curvilinear eyebrow incision with vertical extension (Conley). This was used largely to insert a plate to repair postoperative deformity.

3. An eyebrow butterfly incision with vertical extension (Hagoman).

4. The eyebrow butterfly incision bilaterally, without extension vertically (Hauley).

Any of these last three incisions should give adequate exposure, when indicated, either for osteoperiosteal flap or for insertion of a metal plate to repair postoperative defects.

5. One other is mentioned, a horizontal transfrontal incision (Durling), mainly of value if the tumor appears to be attached in the area of the frontal sinus septum.

When the anterior sinus wall is removed, in case of a large tumor, the subsequent cosmetic result may be quite undesirable. In Case 1, the author used the typical Killian incision and removed the anterior plate of the right side, and to a moderate extent over to the left of the septum, so as fully to expose the tumor and to permit easier surgical attack. Even the right supraorbital ridge was so involved as to necessitate its sacrifice.

Twenty-two years later (Case 2) an attempt was made to prevent deformity by the *lid operation*, and in Case 3 a further improvement in technique was devised by using the flap or *hinge operation*. Illustrations of these three techniques, photographs and a moving picture film of Case 3 will be shown.

SIX CASES OF FRONTAL OSTEOMATA (EPITOMIZED).

Each showing a clear-cut difference in its pathological, clinical and surgical details.

Case 1: M. J. Surgery was performed in August, 1928 at the Battle Creek (Mich.) Sanitarium, with the assistance of Dr. C. G. Wencke. A large part of the anterior frontal plate and most of the right supraorbital ridge was removed, with resulting marked deformity.

Case 2: E. J. Operation performed on July 2, 1950, at the Glendale (Calif.) Sanitarium and Hospital. This patient was referred to the author by Dr. Leland R. House, who was unable to operate, due to an acute injury to his hand but was present as observer and adviser. With the assistance of Dr. Albert J. Patt, the anterior frontal plate was removed, entirely and preserved as a lid to be replaced at the conclusion of the tumor removal.

Case 3: C. B. Surgery was performed on Dec. 4, 1950, at White Memorial Hospital. This patient was referred to the author by Dr. H. James Hara, who collaborated, and with the assistance of Dr. George Weyand, resident, a new technique was utilized, making a hinge of the frontal plate, base downward, and, thus, preserving a great part of the circulatory supply.

Case 4: M. S. This operation was performed by Dr. Leland R. House at the Los Angeles County General Hospital, June 9, 1949. It is used in this presentation by his consent and by permission of the General Hospital authorities, to illustrate a relatively small tumor, with clinical symptoms, relieved by surgery.

Case 5: E. H. This patient was referred to Dr. Leland R. House by Dr. A. L. Muff, for consultation and advice. By permission of Dr. House, the patient is presented in this series to illustrate a very small, and still silent, tumor in a very small frontal sinus, if indeed it really is frontal. The disproportion portends the necessity for relatively early removal of the tumor.

Case 6: A. R. This patient was operated upon by Dr. Wm. T. Grant, neurological surgeon, White Memorial Hospital, on Feb. 26, 1948. It illustrates certain possibilities of intracranial complications, in neglected cases, and the grave operative procedures thus necessitated.

None of these six cases has been previously reported.

PRESENTATION OF THE SIX CASES OF OSTEOMA OF THE FRONTAL SINUS IN DETAIL.

Case 1: August, 1928. Martin J., a sturdy sheet metal worker, age 30 years. His complaint was a bulging of the right eye, with constant diplopia. This had been observed several months, gradualy getting worse. There were no other symptoms. A tentative diagnosis of a neoplasm or mucocele behind the bulb was determined by X-ray study to be an osteoma of the right frontal sinus. This was confirmed by surgery. Incidentally, the mucocele was also found at this time. The procedure was performed under local anesthesia. The sphenopalatine area, the nasofrontal region and the middle meatus were packed with cocaine mud. A solution of novocaine with suprarenin was used to infiltrate the supraorbital region, blocking both the supraorbital nerves.

An incision was made beginning at the external end of the right eyebrow and through it to the nasal angle. This was extended down the side of the nose, across the bridge, up the left side of the nose and as far through the left eyebrow as the supraorbital notch.

An attempt was made to spare the anterior sinus plate, but this was not possible, as the tumor was found to completely fill the sinus, and, therefore, was lying immediately under the outer table. In the complete exposure of the tumor, which weighed 12.8 gm., it was necessary to remove all of the anterior wall of the right sinus and a portion of the anterior wall of the left. The septum between the two sinuses was practically destroyed by the pressure of the growth.

In order to remove the tumor, the right supraorbital ridge had to be sacrificed as certain tooth-like processes of the neoplasm had penetrated the floor of the sinus so close to the ridge that separation and conserva-

tion were not possible. The anterior ethmoid region contained another tooth-like projection about 1 cm. in diameter. The origin of the tumor was in the region of the right nasofrontal duct. Early in the surgical attack an attempt was made to bite off a portion of the upper edge of the tumor. A large mastoid rongeur was used, but under forcible closure of the handles the instrument broke, revealing the intense hardness of the bony mass. In exploring downward around the growth, an area was reached that was as surprisingly soft, having the consistency of maple sugar. A furrow was made in this soft bony tissue about the periphery of the hard growth. The osteoma was then easily rocked off and removed. This soft base was entirely cleaned away, down to hard, normal appearing bone.

In 1880, H. Knapp stated that one chisels around the base of the osteoma, "not attacking it from the surface or chiselling or sawing it off piecemeal."[11]

In 1892, Edward Jackson emphasized the "value of the drill when skillfully used, in effecting safe removal of part of bony growth where the base has to be left."[11] He refers, no doubt, to the sessile soft growths.

The right frontal duct was well opened. There was no mucosa evident in the right sinus. In the orbit, however, there were several polypi and a quantity of mucocele material. The mucosa of the left frontal sinus was also polypoid and was completely removed. The left nasofrontal duct was not touched. The remnant of the septum between the sinuses was removed down to the base so as to insure drainage of both sinuses through either side. Two small fenestrated rubber tubes were passed down to the nasal vestibule through the right nasofrontal duct. One tube extended upward and outward to the outer corner of the excavation made by the tumor. The upper end of the other tube lay free in the right frontal sinus. The tubes were gradually removed beginning on the third day. Surgical recovery and disappearance of the protrusion of the eye and of the diplopia ensued in a short time.

The patient valued the tumor so highly that he kept it in a bottle with alcohol. He returned to the clinic every few months to have the "alcohol changed." His wife recognized his evaluation of the tumor so well that when he died of pneumonia, in 1943, after 15 years, she placed the tumor in the casket with him; therefore, it cannot be demonstrated at this time.

The postoperative facial deformity was marked. Today it, no doubt, would be overcome by a tantalum implant; however, the defect did not seem to be particularly disturbing to the patient nor to his family.

Case 2: Mr. Elwyn J., age 76, was referred to Dr. Leland R. House by Dr. A. L. Muff, May 29, 1950. The patient had severe pain in the right frontal region and a somewhat lesser disturbance of the entire right half of the head. No evidence of sinusitis was found by a consultant at the time the pain began. The patient noticed a swelling over the right eye and that the skin in the same area was very sensitive, soft and yielding, not bony hard. In recent days he had developed a low grade fever. Penicillin seemed to afford some relief. Dr. Muff had an X-ray study of the sinuses made on May 28. This showed a density in the right frontal sinus which was diagnosed as a bony growth.

The patient first presented himself in person on July 2. Study of the accompanying film led to the confirmation of the diagnosis of a growth,

specifically an osteoma. The tender area of the skin was soft and evidently meant an area of erosion of the anterior sinus wall.

Appointment was made for surgery for the morning of July 24. The evening previous, Dr. House suffered an incapacitating injury to his right hand. The author was asked to take charge and to conduct the operation. Dr. A. J. Patt and Dr. George Weyand acted as assistants. Dr. Colver, recalling the experience with Case 1, proposed to remove the anterior sinus wall entirely as a cover or lid, preserve it in hot saline and replace it after the tumor was removed. There was a question whether the bone would "take," but it was felt that nothing was risked in making the attempt. Dr. Albert J. Patt, following the measurements of a tracing from the X-ray film, made an incision through the soft tissues and periosteum about the periphery of the sinus. Next, a series of small drill holes were made through the plate into the sinus. These holes were then connected by a smaller drill, making a perforating furrow. The anterior frontal plate was then removed. Examination of the inner surface showed an area of bone erosion, 12 mm. in diameter. The soft tissue in this area was not normal and was curetted clean to the overlying dermal tissue. The thinned border of the circular eroded area in the bone was then cut back by curette until the thickness and appearance of the new border indicated approximate normality. The plate was replaced as a lid, and the periosteum was joined around the whole border with closely placed catgut sutures.

About a month later the whole area began to be painful, red, swollen and very tender. It was feared that the bone was breaking down, until a fenestrated soft rubber tube was introduced from the lower border of the incision. Considerable pus was evacuated, but in the course of a few days all inflammation subsided and the tube was removed. It was evident that the area of traumatic and inflammatory erosion had had very low resistance and had succumbed to the resident bacteria. At no time was the sinus itself or the bone substance, in general, involved in the infection.

On Dec. 26 another X-ray study was made. (There had been two others since the surgery.) The Roentgenologist reported: "There are still some evidences of the bone fragments in the anterior portion of the right frontal sinus. There has, however, been considerable absorption of the bone since the examination of Oct. 26, 1950. The burr holes are still evident in the upper portion of the frontal plate, above the absorbing area of bone.

Conclusions: Continued absorption of the bone fragment forming the anterior wall of the right frontal sinus. Whether or not the remaining bone represents a sequestrum is difficult to say. It probably does, however, and is gradually being absorbed.

The surgeon does not fully concur in this conclusion. Whether the bone will absorb, be thrown off or survive sufficiently to make an efficient lid is still an open question. At present the whole area palpates hard, there being a dimple or retraction of skin over the area of erosion that was present before surgery.

Case 3: During the summer of 1950, Dr. H. James Hara learned of the projected paper on osteoma. This recalled to him a patient that he had had a number of years ago. At that time, in August, 1938, a Mrs. Caroline B. had consulted him in regard to nasal trouble. She was then 47 years of age. She was found by X-ray study to have marked disease of the right maxillary sinus and of the right ethmoid labyrinth. A right

Caldwell-Luc and a transantral ethmoidectomy were performed by Dr. Hara, assisted by Dr. Merrill O. Dart, resident. During the course of the radiological study, there was observed a small osteoma of the left frontal sinus. It measured about 1 cm. or less in diameter and was silent.

In September, 1950, the patient, at this time aged 59, was located and was advised to have another X-ray study. This new film showed that the tumor had grown very considerably, nearly filling the sinus. It appeared to be lobulated, the greatest diameter being 30 mm. The patient desired to have this tumor removed.

After discussing the proposed surgery, involving a modification of technique by Dr. Colver, Dr. Hara agreed to arrange for moving pictures of the operation. After preoperative medication intravenous sodium pentathol anesthesia was induced. The eyelids of the left eye were closed by silk sutures. Novocaine infiltration was induced over the left eyebrow, across the bridge of the nose, and for a short distance along the right eyebrow. A three-quarter butterfly incision was made and the skin elevated as thin as possible, leaving a thick subcutaneous layer of tissue over the periosteum of the frontal plate. Bleeding was controlled by coagulation. The uncovered area was demarked according to the size of the tumor, and the sinus outline, using the X-ray film and a tracing as a guide. About 12 penetrating burr holes were made by Dr. George Weyand up the mesial border, across the superior border and down to the level of the brow along the outer border. The line for the holes was exposed by an incision through the periosteum with minimum retraction. Instead of connecting the holes with drilling as was done in Case 2, they were joined by means of a sharp chisel, loosening, but conserving the bone substance. Then the modification of technique was done by making two short incisions in the supraorbital line. Through these incisions the chisel was insinuated and sharply tapped. This broke or definitely weakened the bone of the plate horizontally, but spared the periosteum and connective tissue and thus the circulation from below. It also insured the integrity of the supraorbital border. Next, an elevator was passed along the line of bone incision on the lateral and upper borders. By gentle loosening and then by prying or lifting, the horizontal line at the brow level was cracked and the frontal plate was swung forward and downward like a door, using the soft tissue as a hinge. The tumor exposure and surgical accessibility were excellent. The tumor sprang from the ethmoid area. It was loosened by chisel and rather gentle tapping. This broke the stalk or base of the growth and it separated easily from its bed. The soft base was adequately curetted. The tumor measured 33 mm. vertically and 43 mm. horizontally.

The right frontal sinus was inspected through a tumor produced perforation of the septum. There was a moderate amount of greenish-gray mucoid contents that was aspirated, emptying the right sinus. There was no pus. The mucosa apparently was normal. The rest of the septum between the sinuses was removed. No rubber tubes were passed and no dilating instrumentation was performed on the normally patulous frontal openings into the nose.

The hinged frontal plate was swung up into place. The lower border of overlying connective tissue seemed normal, the two short incisions not being readily visible. A dozen catgut sutures were used to coapt the periosteal borders. Silk sutures were used to close the skin incisions. A large pressure pad was applied over the left frontal area. Postoperative progress has been uneventful. It is evident that the integrity of the bony cap has been insured through preservation of the circulation from below by the uncut thick connective tissue hinge.

In the 1926 edition of the translated textbook of Prof. Hajek on Nasal Accessory Sinus, there is a section on radical operation by the osteoplastic method. The cases reported were purulent frontal sinusitis and not osteoma. Brieger, and later Shoenborn, were given credit for the first cases well over 50 years ago. The outcome of Brieger's case, unfortunately, was not given. He resected and reimplanted the osteoplastic flap. Shoenborn made a skin-bone flap with the base up. Scanty postoperative history was given. Czerny reported a case with cure of the suppurative sinusitis, effected in six months.

In 1897, Golovine reported three cases with base of the flap downward instead of upward. He considered one patient, at least, as cured after nine months. As to the cosmetic result, he said, "the traces of the operation remain scarcely visible."

Hajek states that "definite judgment regarding the value and duration of healing after the osteoplastic resection can be given only after years." The results in the hands of various surgeons were of varying success. The procedure seems to have fallen largely into disuse. As noted above, no case of osteoma, either with the base of the flap upward or downward, seems to have been handled by this method.

One essential difference between these early flaps and the one used in Case 3 is this: The early flaps were skin-bone. The skin incisions were made immediately to the bone and the whole thickness resected without too much regard as to cosmesis. In Case 3 the skin incisions were carefully made, the skin elevated upward and as thin as practical. The bone flaps were made, preserving as thick a layer of vascular bearing connective tissue as possible. The replacement suturing was in two layers, the bone and connective tissue and the skin.

There are two other cases, loaned to the author by Dr. Leland R. House, that illustrate the possible variations in relative size of the tumor and its containing sinus. They emphasize the necessity of careful clinical consideration in making a decision as to whether and when to advise surgery.

Case 4: Maria S., aet. 65, May 24, 1949. This patient was seen and later operated upon by Dr. Leland R. House, at Los Angeles County General Hospital. The case is presented to illustrate a tumor, relatively small, but demanding surgery to relieve annoying symptoms (local pain and left-sided headache). The X-ray film shows the smallness of the growth with a fairly large sinus.

Surgery was done on June 9, 1949, under local anesthesia. The Lynch type incision was made, the anterior plate opened and partially removed by chisel and drill. No special attempt was made to preserve this wall. The tumor measured 6 mm. in diameter. The base was just medial to the nasofrontal duct.

Surgical convalescence and recovery from the annoying clinical symptom was uneventful. A postoperative film was made and is also reproduced herewith.

Case 5: On Dec. 23, 1950, Dr. Leland R. House had a patient, Mr. Eric H., referred to him for consultation. No clinical details are available. The probability is that the case is symptomless as regards the frontal sinus. The right frontal sinus is so small that it has scarcely invaded the lower part of the frontal bone. Within it is a very small shadow, evidently an early osteoma.

The diameter of the growth is 5 or 6 mm. and the diameter of the sinus in that area is 8 or 9 mm. Here is a problem, indeed. It would seem that in spite of the smallness of the growth and its present silence, the relative absence of space for development would indicate the desirability of keeping early surgery in mind. It is probable that X-ray study at six-month intervals, together with the cooperation of the patient in observing the development of any clinical symptoms, will determine the therapeutic course to pursue. When the tumor impinges against the sinus walls or when disturbing symptoms develop, it is evident that surgical intervention will be clearly indicated.

Case 6: In February, 1948, Mr. A. R., age 30, was studied, by Dr. William T. Grant, neurologically and by X-ray film. A preoperative diagnosis was made of osteoma of the left frontal bone (sinus) and a glioma of the left frontal lobe.

On Feb. 26, surgical approach was made through the left temporofrontal area using a skin-bone osteoplastic flap, base out. The osteoma was removed in fragments. Its base was "in the anterior ethmoidal or in the medial part of the left frontal sinus." The osteoma was irregularly knobby. The posterior sinus plate was largely eroded, and the tumor was firmly attached to the dura "everywhere." The preoperative diagnosis of glioma was changed to a postoperative diagnosis of "cyst of the frontal lobe due to chronic irritation." This cyst was very large, containing a great quantity of gelatinous, yellowish fluid.

The surgical summary records: Left frontal craniotomy with removal of osteoma and amputation of left frontal lobe.

The history relates as a possible etiological factor and the resulting clinical course *1.* a fall with injury to the left forehead 17 years previous. *2.* Some sort of falling five years ago, beginning with severe headaches, nausea, occasional vomiting with drawing of the legs and with unconsciousness for a short while, occurring every two to three months until six weeks ago; and *3.* the past six weeks the attacks have occurred several times a day, the patient being unable to work.

Surgical and symptomatic recovery were so satisfactory that the patient was permitted to return home within a few days; however, about two weeks later his head began to ache rather seriously. When this information was transmitted, the surgeon advised return to the hospital at once. This advice was not followed until two weeks later. The patient expired while enroute. Postmortem revealed an abscess in the remnant of the left frontal lobe that might have been drained and the case completely restored. Thus, again, a lesson of neglect and delay.

Each of the six cases herein reported is of the pedunculated, hard bone variety. Each one illustrates definitely different characteristics of osteoma, but there is one type of frontal sinus growth that is not represented. This is the sessile or spongious bone mass. The author has never had opportunity to deal with such a growth; however, quite recently, by invitation of Dr. Victor Goodhill, he was given opportunity to observe and to hold various and assorted retractors, when such a case was operated upon, at the Eye and Ear Hospital of Los Angeles, by Dr. Goodhill. The doctor has kindly consented to discuss this paper and to include consideration of the sessile type.

The library research and the preparation of suitable abstracts was done by Dr. Allen G. Thomas, resident in otolaryngology at the Los Angeles County General Hospital. His assistance was invaluable in the preparation of this paper.

Mrs. Ella Crandall, librarian of the medical library of the Los Angeles County General Hospital, extended fine cooperation to Dr. Thomas and to the author, which was greatly appreciated.

The Department of Visual Education, C. M. E., under its head, Edward N. Hamilton, produced the moving picture film that so clearly indicates the technique of the new operation that was used in Case 3; also, the photographs and the lantern slides. Thanks are due to Mr. Hamilton and to his associates for their ability and cooperation.

Also, appreciation is extended to Lucile C. Innes for the illustrations of the surgical approach and technique used in Cases 1, 2 and 3.

And lastly, deep appreciation is due to the various colleagues who have permitted the inclusion of their patients' clinical records and X-ray films to make possible the presentation of this series of six hitherto not reported and so diverse cases.

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SOME LESIONS OF BRONCHI AND ESOPHAGUS.*

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We hope this paper will be of interest, not because of its originality but because it represents what we are doing as a practicing otolaryngologist and a practicing surgeon in a small to medium sized city. This paper represents a joint effort, I presenting the endoscopic side, and Dr. Chunn the surgical picture.

The growing practice of thoracic surgeons to do their own endoscopy, we feel, is disadvantageous to the smaller communities, because it does not allow the endoscopist the necessary volume to insure the proficiency necessary to handle the diagnostic and foreign body work of their communities. Most of these cases are seen first by internists and general practitioners before being seen by us. The patient presents himself because of cough, shortness of breath, difficulty in swallowing or the production of purulent and bloody sputum, persistent thoracic and retrosternal discomfort. The tools at our disposal for diagnosis are the common ones — history, X-ray, both plain and with contrast media and endoscopy.

Bronchiectasis is the most common condition seen by us; that is, acquired, not congenital, bronchiectasis. History, examination, X-ray, bronchogram and bronchoscopy show us the extent of the lesion. Whenever possible, surgical removal is the treatment of choice. In these pitiable cases where surgery is not practical medical treatment is indicated. The use of chemotherapy, antibiotics and vaccine has been rather disappointing. We find that time-honored postural drainage together with bronchial aspiration or lavage offers the great-

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est help. I would like to recommend the more widespread use of bronchial lavage as an office treatment in this condition. We use a Coude catheter, and after lightly anesthetizing the nostrils and pharynx introduce the catheter into the trachea, and with a large metal syringe instill 40 to 60 cc. of hypertonic saline, as Bledsoe's solution. We have also used solution of penicillin-tyrothecin. The patients relieve themselves of a vast quantity of purulent material by coughing. This treatment will enable many to lead a useful life who would otherwise be complete invalids.

Malignant disease of the lung, particularly bronchogenic carcinoma, is being seen with ever-increasing frequency. This increase may be due to the more thorough study given patients with chest complaints, the more frequent X-ray of chests by private practitioners, and mass X-ray examinations by the health department.

Diagnosis of pulmonary tumors is one of the most difficult. Its early symptoms are not significant. Clinical experience and the constant awareness of the cancer danger is the greatest factor in the early diagnosis. Early diagnosis is imperative for curative surgery. One may not wait for positive bronchoscopic or positive cell findings in bronchial washings. Suspicious X-rays and suspicious symptoms may warrant an exploratory thoracotomy in the face of otherwise negative findings. This does not mean that a careful endoscopy is not necessary even though negative. To check the condition of the tracheobronchial tree prior to surgery is a must. While it is true that most cases where X-rays are strongly suggestive, and a biopsy is readily obtainable by bronchoscopy, are usually too far gone for curative surgery, this is not always the case. At present we have in the hospital a patient whom I felt was probably inoperable due to fixation of the left vocal cord. Through an exploratory thoracotomy the lesion was found to be removable, and we have expectation of a cure.

Purulent tracheobronchitis, particularly in small children and infants, in our opinion, is too infrequently treated

endoscopically. The gentle bronchoscopic aspiration of these patients frequently will prevent the necessity of tracheotomy and often is life-saving. It is also of great value to culture the aspirated material and test the organisms' sensitivity to antibiotics for best selection of an effective chemotherapeutic agent.

We, unfortunately, have had too little opportunity to treat asthmatics endoscopically to be able to make a critical evaluation.

Tubercular lesions are seen only diagnostically as their further treatment is carried out in sanitoriums.

The modern chest surgeon now offers much help and hope to a large class of patients who in the past have been largely neglected: those having esophageal lesions.

Congenital lesions are not too common; we have seen about three in the past two years. The congenital stenosis offers highest hope of cure; the fistula cases, as a rule, do not survive.

Diverticula is diagnosed by history, X-ray and esophagoscopy and are usually handled by surgical excision.

Foreign bodies like those of the bronchi are individual mechanical problems, and their variety is endless.

The lye burns are still seen too frequently. We follow a routine similar to that of Fisher and Hicks in the handling of early cases:

1. Wash stomach with mild acetic acid.
2. Tube feed if necessary.
3. Esophagoscope in four to five days to determine extent of lesion.
4. Do a gastrostomy and retrograde dilation of lesion, if the lesion is extensive.
5. Use Hurst's bougies if lesion is slight.

Most out of town cases are presented to us in a most unfortunate condition — emaciated, dehydrated and poor surgical risks. We treat them intravenously with blood or fluids, perform a gastrostomy, and start retrograde dilation as soon as practical.

Cardiospasm or achalasia is another obstructive lesion which may require the service of a chest surgeon. Most cases, however, respond to some form of dilation, either the hydrostatic or the Hurst bougie. We have several patients with their own bougies, using them as necessary.

Malignant disease of the esophagus is not too uncommon. All patients with persistent retrosternal discomfort and dysphagia should have contrast medium studies and esophagoscopy. Surgery offers these patients palliation and frequently cures.

I should like to say in closing my part of this presentation that I feel that the otolaryngologist doing endoscopy in the smaller cities has a responsibility which must not be shirked, in ever re-emphasizing to his fellow practitioners in his own and in dependent communities, particularly to the general practitioner, the need for the careful examination by every possible means of the "little cough," or the "little heart burn," and the "mild retrosternal pain." It is in that way only that we can hope to raise the statistical average of cures from malignant diseases of air and food passages.

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The 1951-1952 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on Sept. 1 and continue for a period of 10 months. Registrations must be completed before Aug. 15. Detailed information and application forms may be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minn.

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April 1, 1951.

Audivox Model Super 67.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303. (See Silver Micronic.)
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model 45.
Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.
Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-1 (Whisperwate).
Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL (Standard); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).
Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet.
Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)
Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.
(See Micronic.)

Silvertone Model 103BM.
Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.
Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model M-35.
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Distributor: Sears-Roebuck Co., 925 S. Homan Ave., Chicago, 7, Ill.

Silvertone Model P-15.
Manufacturer: W. E. Johnston Mfg. Co., 708W. 40th St., Minneapolis, Minn.
Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.
Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.
Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 1700.
Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Trimm Vacuum Tube No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Zenith Model 75; Zenith Miniature 75.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

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Aurex (Semi-Portable).
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Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

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Secretary-Treasurer: Dr. Joseph Hampsey, 806 May Bldg., Pittsburgh 22, Pa.

Meeting: Chicago, Ill., Oct. 19, 1951.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Jose Gros.

Meeting: Third Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology, Havana, Cuba, January, 1952. For information write Dr. Chevalier L. Jackson, 3401 North Broad St., Philadelphia 40, Pa.

SECOND LATIN AMERICAN CONGRESS OF OTO-RHINO-LARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

President: Prof. A. dePaula Santos.

Secretaries: Dr. Jose deRezende Barbosa and Dr. Plinio de Mattos Barretto, Hospital das Clinicas, Sao Paulo, Brazil.

Time and Place: Sao Paulo, Brazil, July, 1951.

AMERICAN BRONCHO-ESOPHAGEAL ASSOCIATION.

President: Dr. LeRoy A. Schall, 243 Charles St., Boston, Mass.

Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1, Md.

Meeting: Hotel Claridge, Atlantic City, N. J., May 7-8, 1951, P. M.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Alden H. Miller.

Secretary-Treasurer: Dr. Victor Goodhill.

Chairman of Section on Ophthalmology: Dr. Dennis V. Smith.

Secretary of Section on Ophthalmology: Dr. Carroll McCoy.

Chairman of Section on Otolaryngology: Dr. Howard P. House.

Secretary of Section on Otolaryngology: Dr. Edwin Scobee.

Place: Los Angeles County Medical Association Building, 1925 Wilshire Blvd., Los Angeles, Calif.

Time: 6:00 P.M., fourth Monday of each month from September to May, inclusive.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

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NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. G. M. Billings, Morganton, N. C.

Secretary and Treasurer: Dr. MacLean B. Leath, High Point, N. C.

Time and Place: Sept. 11-13, Hendersonville, N. C. Jointly with the South Carolina Society of Ophthalmology and Otolaryngology.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

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Meeting: Empress Hotel, Victoria, B. C., May 27-31, 1951.

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